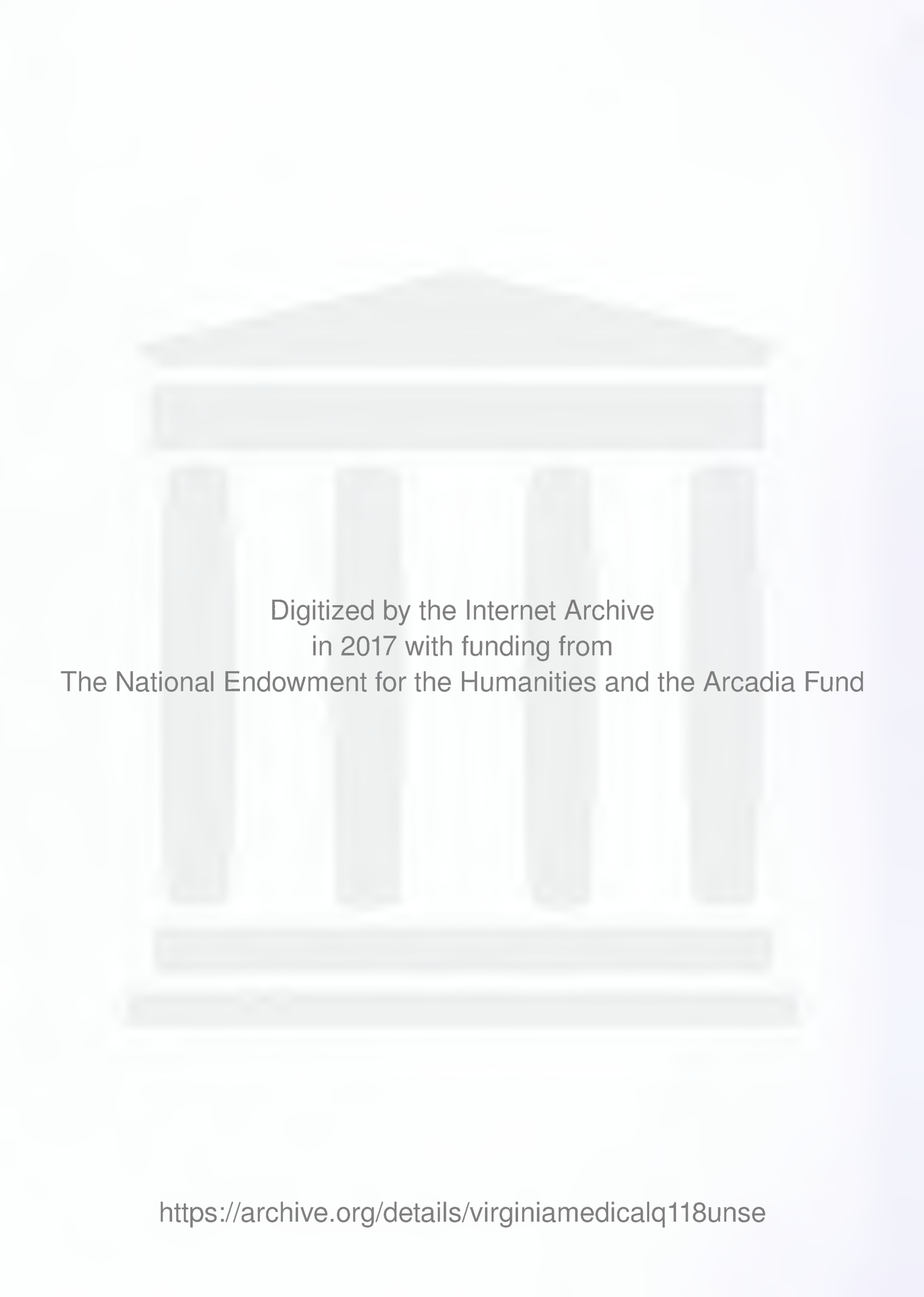

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VIRGINIA MEDICAL QUARTERLY

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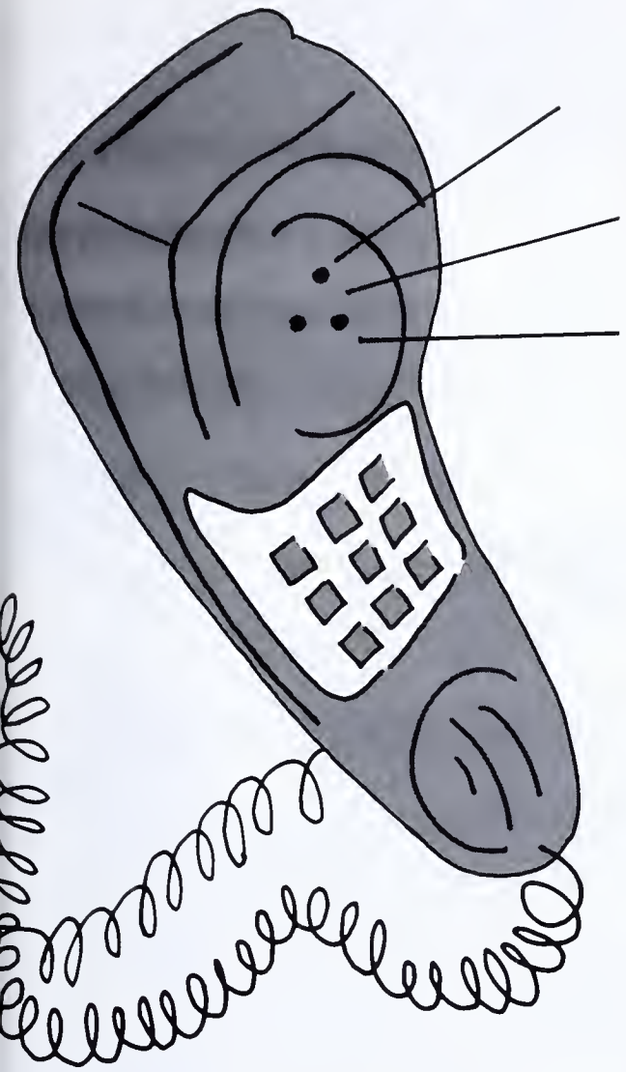

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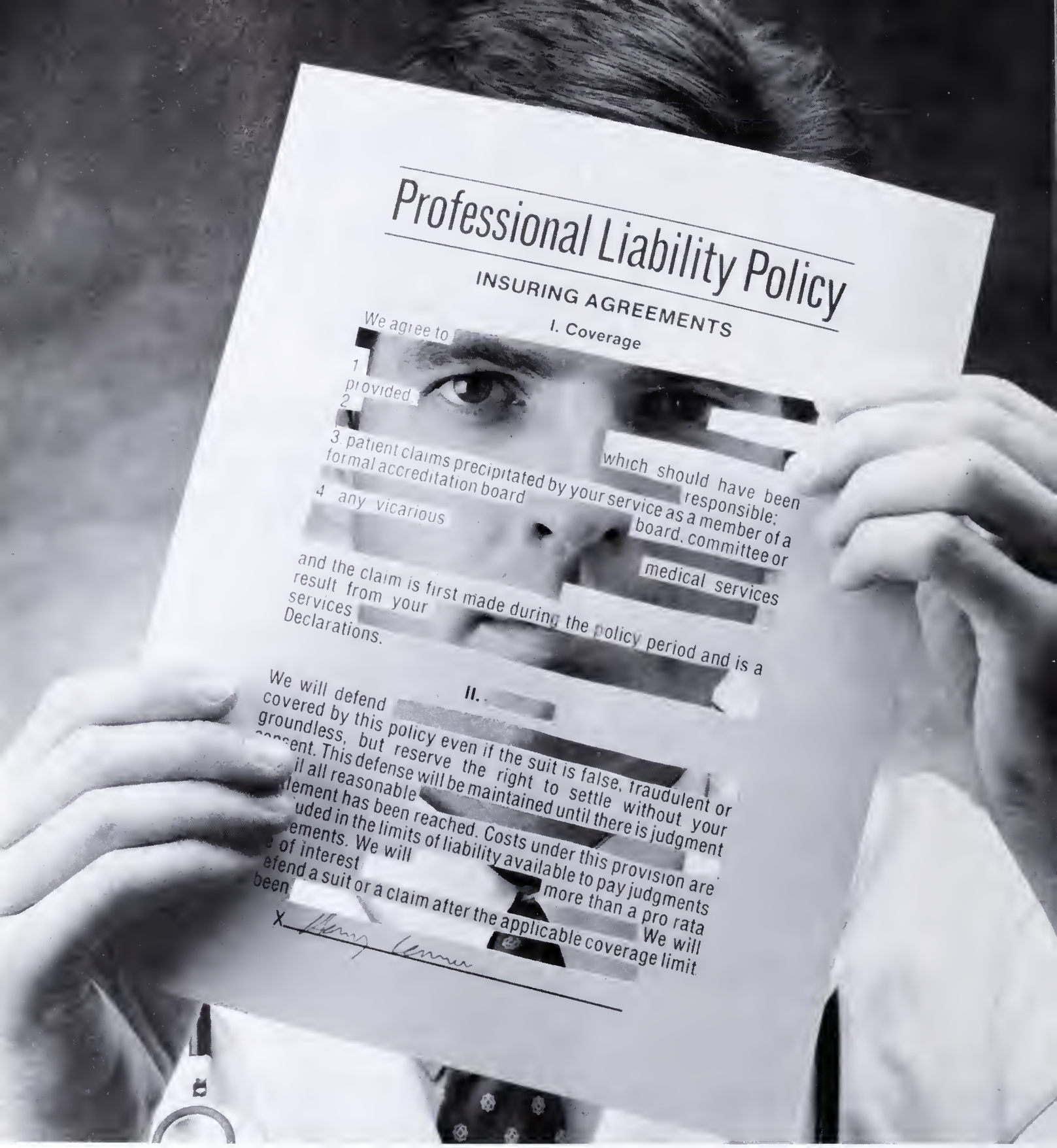

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LETTERS

We welcome your Letters to the Editor.
All letters should be sent to the Editor, VIRGINIA MEDICAL QUARTERLY, 4205 Dover Road, Richmond VA 23221. Please include your name and telephone number. VMQ reserves the right to edit all letters.

Professional courtesy defrauds, say health insurers

One of my associates recently showed me a letter from an insurance company advising us that the practice of giving professional courtesy to fellow doctors and their families could be considered fraud!

Their twisted logic was that since they only paid a percentage of my fee that if I didn't ask for a copayment that my fee wasn't really what I claimed—it was less the copayment I was giving; therefore their percentage should be even less and I was defrauding them.

I have never been billed by the pediatricians who saw my three children, or by the surgeons who repaired my ruptured diverticulum, or by the internists who have cared for my mother. I think we are about to lose something that is very precious.

Jack C. Turner, MD
425 Hawthorne Drive
Danville VA 24541

Note: See Dr. Kendig's editorial on page 53.

He likes idea of return to medical school for CME

I enjoyed reading Dr. David Zohn's point of view on the downside of compulsory medical education in the autumn issue of VIRGINIA MEDICAL QUARTERLY.[1] Here in Michigan we have state-prescribed hours to fulfill; at one time we had to have our signatures on the forms "certified," but I resisted that some ten years ago and a year later this demeaning item was dropped. I could not, however, talk the Michigan State Medical Society into resisting "commanded medical education," as it has been termed. It was felt such effort would stem malpractice problems. Nuff said. One of the best internal medicine courses I ever had the privilege to attend carried no CME credit! This at Henry Ford Hospital, Detroit.

Dr. Zohn's solution, for a return to medical school every ten years for three weeks or so of renewal, is an interesting and very good idea, but it ought to have flexibility built in, e.g., 10th anniversary = 10 ± two yrs. Various demands upon one's time at a given moment could make an exact decade renewal burdensome, even for so short a time as three weeks. Some might opt for one week at a time over three years or some other variation. So be it. The state should have nothing to do with its supervisional aspects nor should federal bureaucrats.

Despite having been in Michigan for some 20 years, I still get VIRGINIA MEDICAL—interned at MCV in '56, medical staff at U.S. Naval Hospital, Portsmouth, '60-'65, and at UVa '65-'70.

Congratulations on a fine point of view. I agree!

Richard A. Wetzel, MD
Department of Nuclear Medicine
Beaumont Hospital, Royal Oak MI 48073

1. Zohn DA. Compulsory medical education: the downside. Va Med Q 1990;117:368-369

Two see need for change in Va Med Q's focus

We are all overwhelmed with journals and other publications, and in that competition it is difficult for the VIRGINIA MEDICAL QUARTERLY to find its proper place in the sun. There is, however, a niche for general

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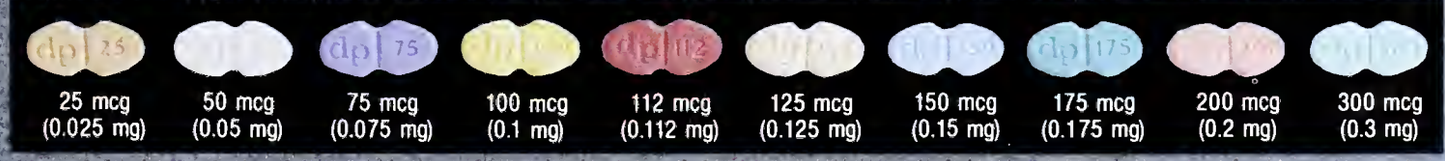

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purpose publication which has not been filled and might conceivably be filled by the QUARTERLY. That is a discussion of health care issues, with particular attention to the point of view of the practicing physician. We are swamped with pronouncements by politicians, labor leaders, business executives, economists and virtually everyone else about what is wrong with medicine and how it can be reformed, but there really is no forum for the practicing physician to have some input in an organized fashion in this clamor of voices. A problem, of course, is how to obtain qualified articles for inclusion. My suggestion would be to consider a foundation. It might be either a foundation established by the Medical Society of Virginia for that purpose or possibly a friendly local foundation which would be interested in aiding this endeavor. At any rate, regularly scheduled symposia might be held, either at large community hospitals or at the various medical schools. These could be on predetermined topics and then edited and published by VIRGINIA MEDICAL. Although outside experts from universities or from all the various other disciplines in medicine might be represented, particular attention might be made to include practicing physicians and have their points of view expressed as well.

Although it is a giant leap, the Medical Society of Virginia and its journal might be to health care and health policy issues what the Massachusetts Medical Society and its organ, the *New England Journal of Medicine*, is to scientific articles. Being so close to Washington, we can make this "must reading" for everybody involved in formulating and delivering health care.

David A. Zohn, MD

1515 Chain Bridge Road, #102
McLean VA 22101

If the comments of colleagues are any indication, I think that the time for a revision of VIRGINIA MEDICAL to focus on health care issues is long overdue and would serve a very useful purpose. With every passing day practicing physicians are increasingly overshadowed by an expectation of wrongdoing and increasingly restricted in their freedom to exercise the clinical judgment and skills they worked so long and hard to acquire. It is clear that one of the primary reasons for loss of support for the American Medical Association as demonstrated by the issue of unified membership is the growing perception that its leaders lack the conviction and willpower to defend the physicians they are supposed to be representing. It seems to me that a publication devoted to the discussion of health care issues would be an expression of concern on the part of the Medical Society of Virginia for its members that is so sadly lacking on a national level.

Donald D. Haut, MD

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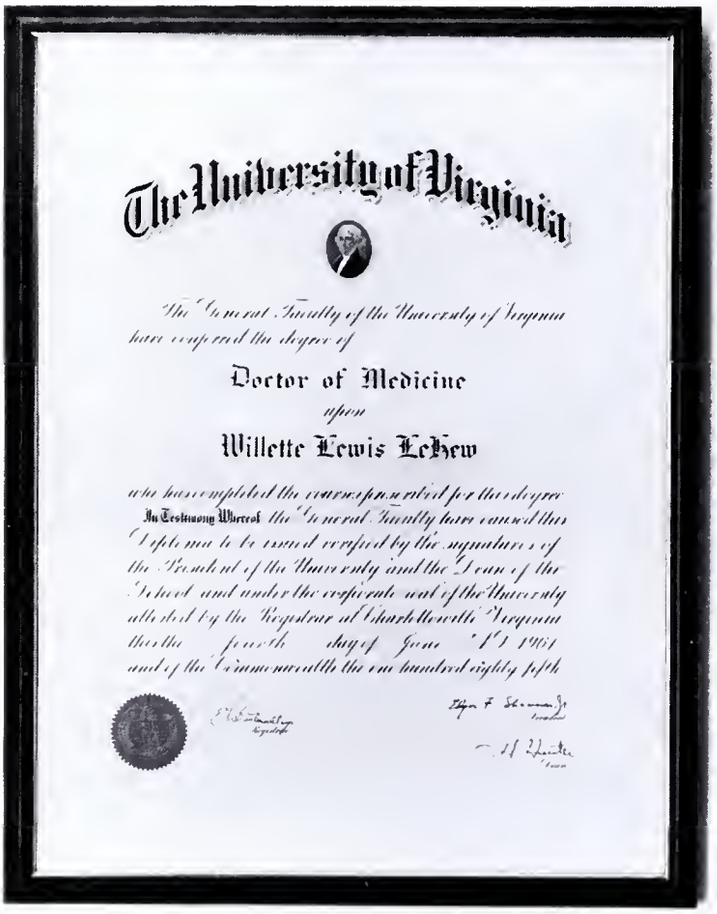

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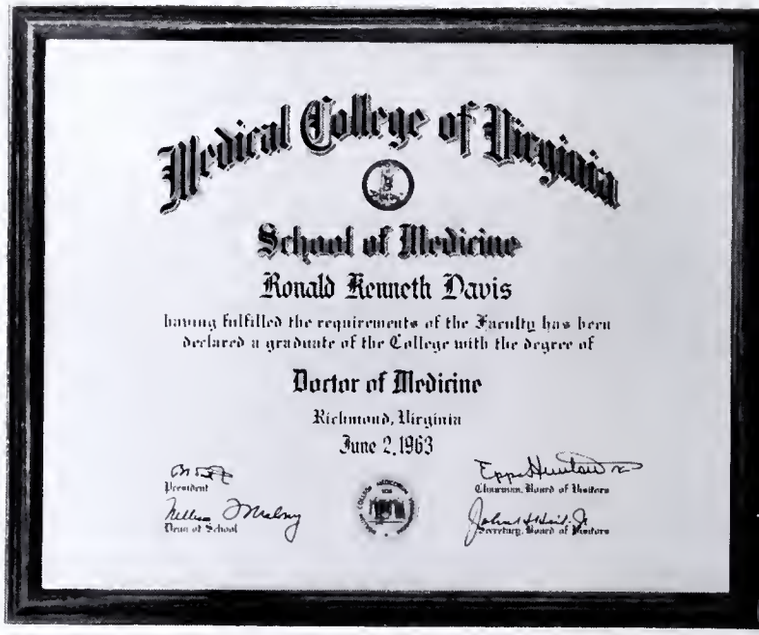

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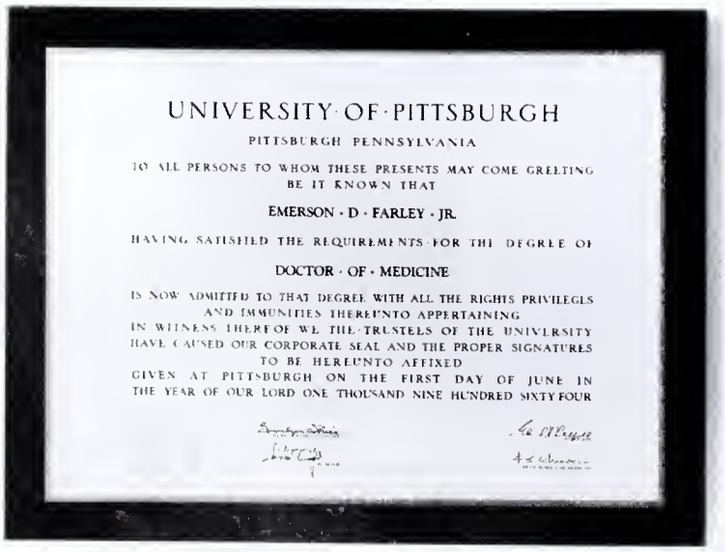

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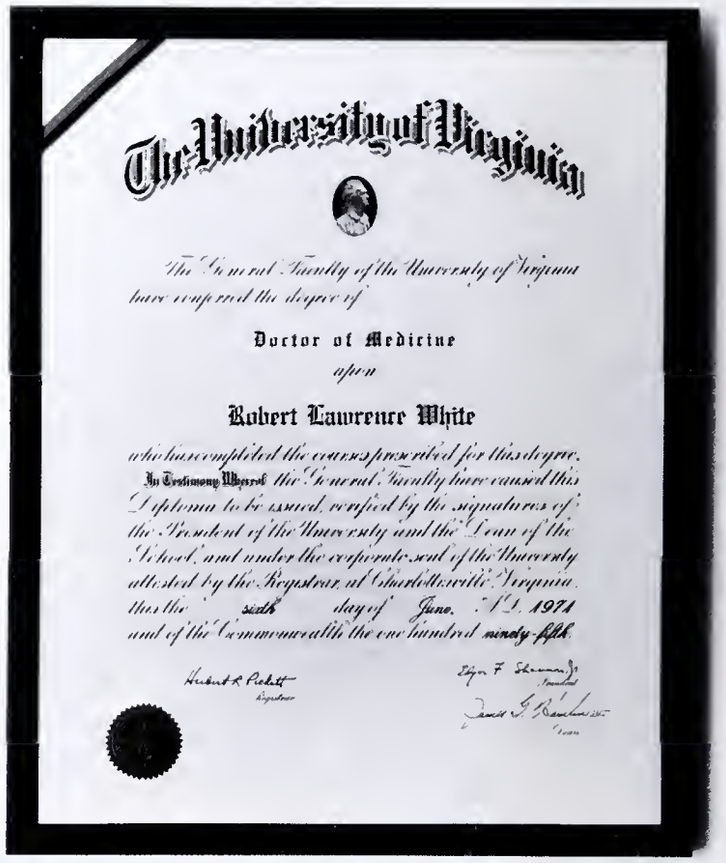

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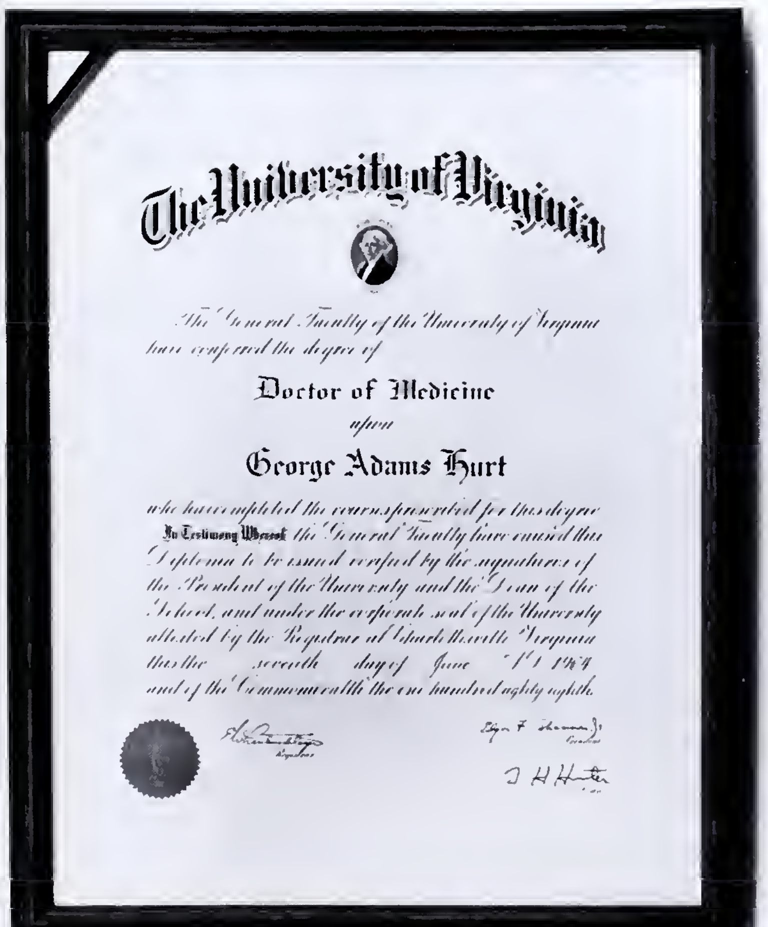

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Shown in the lobby of the Homestead following their installation at the annual meeting are, from left, Dr. John A. Owen, Jr., Charlottesville, President; Dr. John W. Hollowell, Portsmouth, President Elect; Dr. Carol S. Shapiro, Woodbridge, Second Vice President; Dr. Clarence A. Holland, Virginia Beach, First Vice President; Dr. Lawrence K. Monahan, Roanoke, Vice Speaker, and Dr. George E. Broman, Culpeper, Speaker. The new Third Vice President, Dr. W. Kenneth Blaylock, Richmond, was not present for this picture.

Testimony to an Abiding Faith

Presented before the Medical Society of Virginia's House of Delegates at the 143rd annual meeting on November 3 at the Homestead in Hot Springs after Dr. Owen's installation as President.

The text for my sermon today is from the 25th chapter of Matthew, verse 35: "I was a stranger, and ye took me in."

When I came to the University of Virginia in 1960 and joined the Albemarle County Medical Society, I was assured by one and all that a faculty member would find himself a very uncomfortable fish-out-of-water at any Medical Society of Virginia meetings. So in 1974, when I made a presentation to the Council, I quaked in my boots. But the Council, although it voted down my proposal, heard me with courtesy and attention and encouraged me to keep coming back. That's the sort of thing that's been happening to me over and over ever since.

Those who disparage medical "politics" have never grasped the secrets of its appeal: the intrinsic value of a vote, the self-abnegating trust and comradeship of a coalition, and the absolute inviolate sanctity of an honor-bound commitment. These are no light matters, and college Honor Systems can take comfort in the reality of their real-world counterparts. Without an abiding faith in these things I would not be here today.

Our tradition in the Medical Society of Virginia is to hold the election first, then the campaign promises. There is much I want to do—and with your help will do—during the coming year.

First, we need to give attention to our infrastructure, at four different levels: 1) We need to continue to add new members, with special attention to unification dropouts, medical students, house officers, young physicians, and other newly licensed physicians. 2) We need a careful, objective survey of our depleted, overworked staff, enabling us to strengthen it for new tasks, 3) We need clearer direction for and greater vigor within our numerous committees, And 4) the Council needs to work harder as a two-way channel of communication between the membership and the leadership.

Second, I pledge to work hard this year on all aspects of our legislative program. We have reshaped and refocused our lobbying team and I want to be a part of that team, learning as I go how best to articulate our basic position that what's best for the patient is what's best for the Medical Society of Virginia. I will need your help there also. Let's begin by your marking off January 16, 1991, on your calendars as "Legislature Day". Heidi Guerrero, president of our Auxiliary, has planned an audacious takeover of the General Assembly building on that day and she needs YOU to serve on her shock troops!

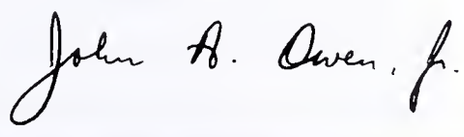
Seriously, I commend to you the philosophy that our General Counsel, Sandra Kramer, brings to the lobbying effort: that the most effective lobbyist is not one who browbeats or armtwists, but who educates the law-makers on all points of the issues, effecting truly informed legislation. Our goal is to earn from the General Assembly the encomium which Robert E. Lee gave of J. E. B. Stuart: "He never brought me a false piece of information."

Finally, if we grow in numbers, in staff strength, in Council and committee efficiency, and in stature in the eyes of the General Assembly, and yet do nothing positive to improve the health of this and future generations of Virginians, we are become as a sounding brass and tinkling cymbal.

Virginia's Secretary of Health and Human Resources, Howard Cullum, has twice come before us, with plain and honest discussion of Virginia's budgetary problems, openly inviting us to bring to him any ideas or suggestions for solving this manifold problem. Here is a golden opportunity for us to distill all our experience and accumulated wisdom into innovative programs, with the unspoken assurance that the Department of Health and Human Resources will become a willing, even active, partner in them. It follows that our legislative problems must diminish as together we remove the impetus to introduce them. All this shall be as bread cast upon the waters, which will return to us after many days.

So with energy and high hopes, with happiness and humility, and with deep appreciation to those who have helped me thus far, I begin—and now I need your help more than ever, to make this a year of achievement for the Medical Society of Virginia.

And I will never again feel myself a stranger among you.



House of delegates action on resolutions

Here are the results of each of the resolutions/recommendations considered by the Medical Society of Virginia's House of Delegates at the 143rd annual meeting held November 1-3 at the Homestead in Hot Springs. For a copy of the resolutions/recommendations in their entirety, call MSV headquarters, 804-353-2721.

Resolution/Recommendation, Sponsor, Description	Adopted	Amended, Adopted	Rejected
ACCESS/PRIMARY CARE			
Resolution X: Attracting Physicians to Rural Areas (*Virginia Academy of Family Physicians*). Medical Society of Virginia to urge state legislature to consider loan repayment plan to attract FPs to underserved areas.		✓✓	
Resolution Q: Direct Access by Physical Therapists (*Virginia Orthopaedic Society*). Medical Society of Virginia to oppose legislation allowing physical therapists direct access to patients without attending physician's diagnosis prescription.	✓		
Resolution OO: Primary Indigent Care (*Chesapeake Medical Society*). Medical Society of Virginia to support funding for expansion of Chesapeake Coordinated Health and Medical Program 1) to include outreach, acute medications, and specialized testing and 2) as demonstration model of primary care for adult indigent patients through public/private partnership.	✓		
Resolution EE: Medicare Assignment (*Richmond Academy of Medicine*). Medical Society of Virginia, with State Department on Aging, to research feasibility of uniform, statewide voluntary Medicare assignment program.	✓		
Resolution FF: Care of Medically Indigent (*Lynchburg Academy of Medicine*). Medical Society of Virginia to encourage component societies to enlist support of local business firms in providing care for the medically indigent and to sponsor free clinics or health department clinic staffing.	✓		
Recommendations, *Federal Legislative/Activities Committee/Council, Item 10.* Medical Society of Virginia to support the AMA's "Health Access America" plan.	✓		
Recommendations 2, 3 and 4, *State Legislative/Rural Health/Indigent Care Committees.* Medical Society of Virginia to 1) continue to promote tort reform as method of improving access to primary care; 2) encourage component societies to work with local authorities to provide care for the medically indigent; 3) oversee development of voluntary Medicare assignment program.	✓		
COMMUNICATIONS			
Resolution GG: Component Society Communications (*Volker Brandt, MD*). Council to submit list of significant health care issues to component/specialty societies.		✓✓	
Resolution II: Transmission of Information to Component Societies (*Volker Brandt, MD*; **Resolution JJ: Communications Committees for Component Societies** (*Dr. Brandt*); and **Resolution HH: Component Society Opinion on State-Wide Medical Insurance** (*Dr. Brandt*). Council to inform component societies of its actions; component societies to establish communications committees; Council to ask component societies to discuss concept of statewide medical insurance for all Virginians.			✗
EMERGENCY MEDICINE			
Recommendations 1 and 3, *Emergency Medicine Committee.* Medical Society of Virginia to endorse State Board of Health language governing emergency medical services and to continue efforts to resolve problems of indigent care in Virginia.	✓		
Recommendation 2, *Emergency Medicine Committee.* Medical Society of Virginia to establish mechanism for EMS systems in Virginia to implement "do-not-resuscitate" orders and surrogate decision-making instruments.			✗

Resolution/Recommendation, Sponsor, Description	Adopted	Amended, Adopted	Rejected
FEDERAL LEGISLATION			
Resolution G: Patient Transfers (*Virginia Academy of Family Physicians*). Medical Society of Virginia to ask Virginia Hospital Association to monitor impact of COBRA regulations restricting patient transfers and report annually to federal/state legislators.			✗
Resolution KK: Anti-Physician Provisions of COBRA (*Leon I. Block, MD*). Medical Society of Virginia to ask AMA to secure repeal of federal legislation restricting patient transfer.		✓✓	
Resolution H: National Practitioners Data Bank (*Virginia Academy of Family Physicians*). Medical Society of Virginia to oppose Data Bank and support legislation transferring its functions to state boards of medicine and Federation of State Medical Boards.	✓		
Resolution K: Inappropriate Government Procedures, Prosecution, and Interference with Practice of Medicine (*Alexandria Medical Society*). Medical Society of Virginia to call for resignation of Inspector General Richard Kusserow, appointment of special prosecutor to investigate, request letter of apology from HHR.	✓		
HIGHWAY SAFETY			
Resolution T: Drunk Driving (*Norfolk Academy of Medicine*). Medical Society of Virginia to urge legislature to mandate license revocation for repeated DUI offenses and eliminate sale of alcoholic beverages by gasoline retailers.	✓		
Recommendations 1 and 3, *Highway Safety Committee*. Medical Society of Virginia to support stronger pedestrian safety laws and legislation prohibiting passengers in bed of pickup truck.	✓		
HIV/AIDS			
Recommendations 1, 2, 3 and 4, *AIDS Committee*. Medical Society of Virginia to encourage participation of component societies/physicians in care of HIV/AIDS patients and in sponsoring community education/information; to continue current policy re testing of HIV patients; and work with state/local governments on HIV/AIDS prevention/care/treatment/ reimbursement plan, with emphasis on pediatric AIDS.		✓✓	
Resolution P: HIV Testing (*Portsmouth Academy of Medicine*). General Assembly to be requested to pass law authorizing physicians to order HIV tests before/during/after surgery.			✗
Resolution O: Pediatric AIDS (*Virginia Chapter, American Academy of Pediatrics*). Medical Society of Virginia to seek legislation for statewide pediatric AIDS committee and pediatric health care teams at each state HIV center.			✗
INTERNAL AFFAIRS			
Recommendations, *Membership Committee*. 1) Continue recruitment efforts, including use of slide presentation; improve outreach for fellows, residents, students; encourage AMA membership. 2) Honorary membership for William H. Barney, MD, MSV President '89-'90.	✓		
Recommendation, *Presidential Address (Dr. Barney)*. Endorse position paper prepared by Executive Committee and VaMPAC's directors reaffirming/strengthening relationship between Medical Society of Virginia and VaMPAC.	✓		
Recommendations, *Bylaws Committee/Committee on Committees/Council, Item 11*. Twelve bylaw revisions to reflect changes in committee structure/nomenclature; one technical correction. Medical Society of Virginia to establish new Ad Hoc Committee on Bioethics.	✓		
Resolution AA: Cardiology Delegate (*Roanoke Academy of Medicine*). Medical Society of Virginia to authorize one delegate from American College of Cardiology, Virginia Chapter, to MSV House of Delegates. Council to consider MSV membership criteria for specialty representation.		✓✓	
Recommendation, *Finance Committee and Council*. 1990-91 Budget, as shown on page 18.[1]		✓✓	

▲ Dr. E. Blackford Noland, Jr., Roanoke

▲ Dr. Michael W. Russell, Culpeper

▲ Four delegates from the Danville-Pittsylvania Medical Society show their appro a House action. From left, Dr. Richard C. Cole, Dr. Jack C. Turner, Dr. Charl Freed, Jr., and Dr. James E. Nevin III.

▲ From left, Dr. William H. Sipe, Newport News; Dr. Hedley N. Mendez III, Poquoson; and Dr. Douglas H. Chessen, Newport News

▲ Dr. James L. Snyder, Clifton Forge

▲ Dr. John A. Hagy, Rocky Mount

▲ Dr. Norris A. Royston, Marshall

Photographs by Werner Gattinger

▲ Dr. Clyde H. Dougherty, Hopewell, and Dr. Carmen R. Rexrode, Blackstone

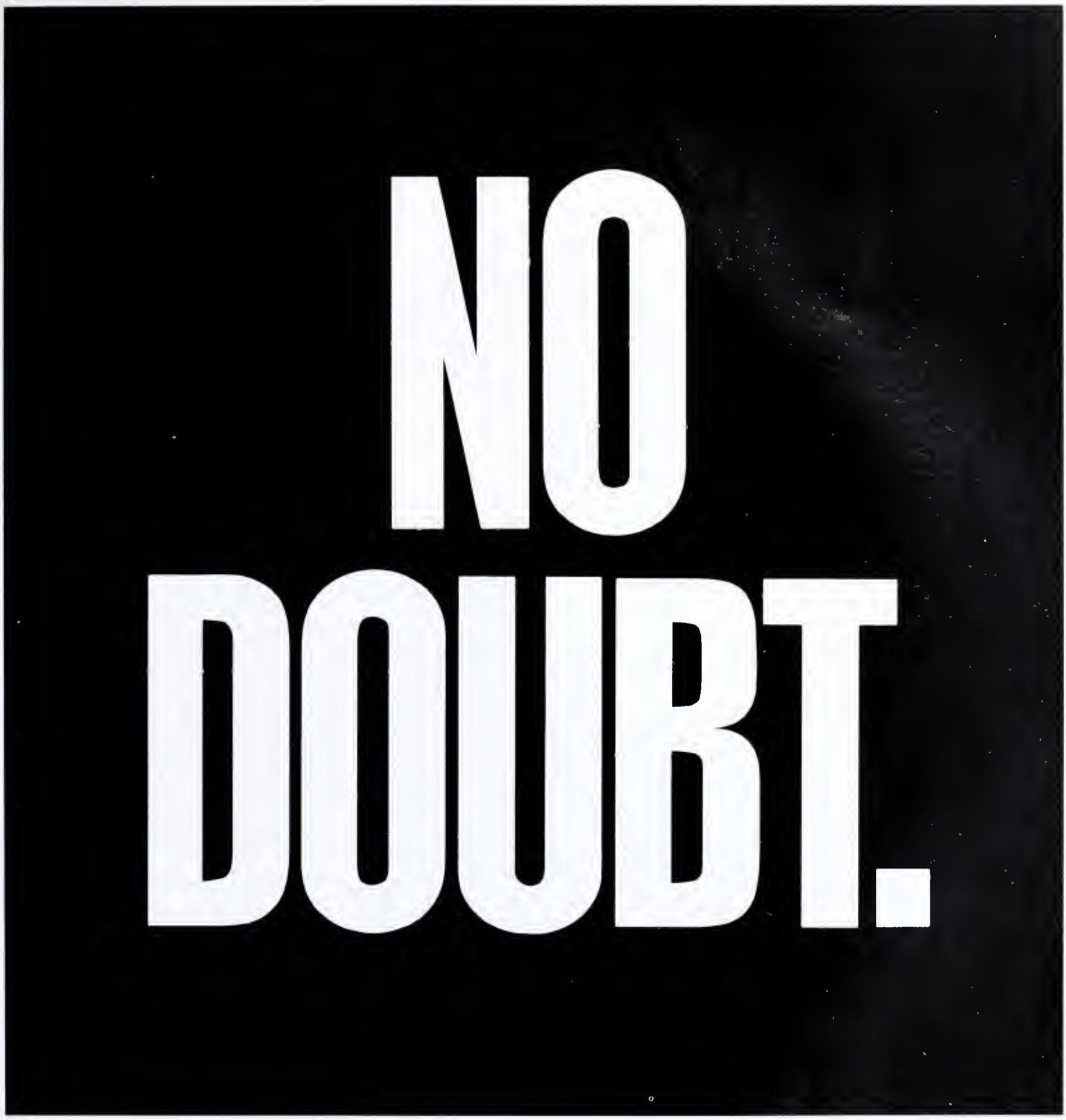
▲ Dr. Robert P. Nirschl, Arlington

▲ From left, Dr. Robert J. Faulconer, Norfolk; and Dr. Colin W. Hamilton and Dr. Mark T. Schreiber of Virginia Beach

Resolution/Recommendation, Sponsor, Description	Adopted	Amended, Adopted	Rejected
Resolution BB: Long-Range Planning (*Richmond Academy of Medicine*). Medical Society of Virginia to implement long-range planning process, utilizing broad membership cross-section, and submit plan to House of Delegates 1991.		✓✓	
Resolution CC: New Advocacy Committee (*Joseph D. Brown, MD*). Medical Society of Virginia to establish Physicians Advocacy Committee for members under investigation by state/federal agencies and hospital committees.		✓✓	
Resolution DD: Education Committee Representation (*C. Robert Meloni, MD*). Medical Society of Virginia to ensure that entities accredited by the Education Committee be fairly represented on the committee and duly informed of its procedures/proceedings.		✓✓	
Recommendation, *Physicians Health/Effectiveness Committee*. Budget of $23,150.	✓		
LIABILITY/INSURANCE REFORM			
Recommendations, *State Legislative/Professional Liability Committees*. Medical Society of Virginia to consider development of fund for patients inadequately compensated under medical malpractice cap and, if findings are positive, to pursue implementing legislation.	✓		
Resolution M: Expanded Malpractice Cap (*Virginia Academy of Family Physicians*). Medical Society of Virginia to oppose proposed extension of current cap to $1 million per defendant.		✓✓	
Resolution N: Malpractice Reform (*Robert P. Nirschl, MD*). Medical Society of Virginia to explore additional types of malpractice reform, including capped binding arbitration.		✓✓	
Resolution Y: Injured Infants Act Coverage for Residents/Faculty (*Virginia Academy of Family Physicians*). Medical Society of Virginia to support either changing Injured Infants Act to waive $5000 annual participation fee for family practice/obstetrics residents and full-time faculty or establishing fund to assume cost of their participation.		✓✓	
Resolution MM: Tort Reform (*Norfolk Academy of Medicine*). MSV Professional Liability Committee to consider the Harvard Study on the New York State medical liability system.		✓✓	
MEDICAL EDUCATION			
Resolution J: Student Loans (*Norfolk Academy of Medicine*). Medical Society of Virginia to urge congressional delegation to extend deferment period of Title IV loans to two years after completion of training.		✓✓	

[1]MEDICAL SOCIETY OF VIRGINIA BUDGET 1990-1991

Salaries	$657,000
Stationery and Supplies	15,000
Office Supplies/Expense, 700 E. Main St.	37,000
Office Equipment: Repairs/Replacement	5,000
Building Expense	5,000
Building Maintenance/Repairs/Improvements	46,500
Telephone	17,000
Postage	30,000
Convention Expense	40,000
Council/Committee Expense	32,000
Delegates to American Medical Association	60,000
AMA Campaigns	25,000
Staff Travel—AMA	6,000
President's Expense	14,000
Presidential Expense Allowance	15,000
President Elect Expense Allowance	3,000
Staff Travel Expense	9,000
Preparation/Distribution VIRGINIA MEDICAL	45,000
Preparation/Distribution DIRECTORY VIRGINIA PHYSICIANS	20,000
Editor, VIRGINIA MEDICAL	2,400
Legal Expense	75,000
Legislative Program	50,000
Walter Reed Commission	1,000
MSV Auxiliary	7,500
Membership Dues, Affiliated Agencies	1,200
Special Appropriations	
AMA/ERF	1,000
Scholarships	
Medical College of Virginia	1,000
University of Virginia	1,000
Eastern Virginia Medical School	1,000
Rural Health	1,000
Student Medical Societies	
Medical College of Virginia	4,000
University of Virginia	4,000
Eastern Virginia Medical School	4,000
Virginia Health Council	2,000
Hospital Resident Section	1,500
Other Special Appropriations	12,000
Insurance Premiums	17,000
Newsletter	30,000
Employee Benefits	
Retirement Fund	160,000
Blue Cross/Blue Shield/Life/Disability	45,000
Payroll Taxes	45,000
Continuing Medical Education	21,500
Consulting Services	
Legislative/Public Relations	79,000
Insurance Actuary	10,000
Physicians' Health/Effectiveness Program	23,150
Part-Time Medical Director	35,000
Miscellaneous	5,000
Computer Operation/Maintenance	7,500
Audit	20,000
TOTAL	$ 1,749,350



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(804) 285-4131

Resolution/Recommendation, Sponsor, Description	Adopted	Amended, Adopted	Rejected
Resolution NN: Student Loans (*Norfolk Academy of Medicine*). Medical Society of Virginia to support intent of HR747 to restore tax deduction for interest paid on educational loans.	✔		
Recommendation, *State Legislative/Rural Health/Indigent Care Committees.* Medical Society of Virginia to confer with medical schools on increasing primary care specialization.	✔		
Resolution W: Increasing Supply of Primary Care Physicians (*Virginia Academy of Family Physicians*). Medical Society of Virginia to work for legislation to increase number of primary care residencies in Virginia, with reduced state support to those schools not meeting such requirements.			✗
PEER REVIEW			
Resolution I: Sanctioning of Physicians in Residency (*Norfolk Academy of Medicine*). Medical Society of Virginia to oppose inclusion in the peer review process of patient care decisions by resident physicians. Referred to MSVRO Oversight Committee.			
PRESCRIBING/DISPENSING			
Recommendations, *State Legislative Committee/Ad Hoc Committee on Nurse Practitioners.* Medical Society of Virginia to oppose 1) elimination of medical supervision of nurse practitioners; 2) direct reimbursement of nurse practitioners; 3) legislation inhibiting hospitals/others in making staff privilege decisions; 4) HB768, carryover bill authorizing nurse practitioners to prescribe drugs. Complete text printed below.[2]		✔✔	

[2] *The complete text of the adopted resolution on nurse practitioner prescribing is as follows:*

1. That the MSV oppose any attempt to eliminate the requirement for medical supervision of nurse practitioners as not being in the best interest of patient care.

2. That the MSV oppose direct reimbursement of services provided by nurse practitioners.

3. That the MSV oppose any legislation which would inhibit the ability of Virginia hospitals to make decisions regarding hospital privileges for nurse practitioners, as well as all other types of health care providers, on a case by case basis at the hospital level.

4. That it is the policy of the Medical Society of Virginia to oppose HB 768. However, if the General Assembly decides that allowing nurse practitioner limited prescriptive authority will improve access to primary care for indigent patients who receive primary health care from state, federal or local agencies, or nonprofit entities devoted primarily to the provision of health care to indigent populations, an amendment which contains the following essential elements would be the only form of HB 768 that the Society would consider sufficient to provide the minimal safeguards necessary to preserve an adequate quality of care to their patients:

A. Limit any expansion of prescriptive authority for nurse practitioners to nurse practitioners who have experience and training in a primary care specialty, and who provide primary care services for indigent patients who receive primary health care from state, federal or local agencies, or nonprofit entities devoted primarily to the provision of health care to indigent populations;

1. require that the nurse practitioner practice under the supervision and direction of a physician licensed to practice medicine in the Commonwealth, in accordance with a written protocol signed by the nurse practitioner and the physician, and filed with the Board of Medicine;

2. require that such written protocols comply with regulations promulgated by the Board of Medicine in consultation with the Boards of Nursing and Pharmacy; and

3. require that regulations promulgated by the Board of Medicine include, at a minimum, the following items: a) a requirement that the supervising physician retain responsibility for the acts or omissions of the nurse practitioner, b) a requirement that the supervising physician review the medical chart of each patient for whom a nurse practitioner has signed prescriptions within a specified time period, and c) a list of the specific drugs and specific conditions for which the nurse practitioner may sign prescriptions, if authorized by the protocol;

B. That the Medical Society of Virginia support the use of pilot projects as a preferable alternative to statewide application of any legislation expanding the prescriptive authority of nurse practitioners;

C. That the Society recommend inclusion of physicians' assistants with nurse practitioners in any legislation granting limited prescriptive authority to nurse practitioners, subject to the limitations and conditions discussed above; and

D. That the Society seek statutory language in any bill authorizing prescriptive authority for nurse practitioners which provides the supervising physician with immunity from civil liability for any facts or omissions of the nurse practitioner that are beyond the scope of, or not done in accordance with, the written protocol or the verbal or written instructions of the supervising physician, unless the physician knew or should have known of such acts or omissions by the nurse practitioner.

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Resolution/Recommendation, Sponsor, Description	Adopted	Amended, Adopted	Rejected
Resolution U: Health Care Delivery in Underserved Areas (*Roanoke Valley Academy of Medicine*). Although the Medical Society of Virginia is not opposed to use of physician extenders, provided they have appropriate medical supervision, it does oppose granting prescribing powers to nurse practitioners/other physician extenders.		✓✓	
Recommendations, *State Legislative/Therapeutics and Devices Committees*. Medical Society of Virginia to support HB1046, the Medicaid Restrictive Drug Formulary Act passed in 1990, with two provisions: 1) no cumbersome override process, 2) review process to identify physicians who abuse prescriptive powers.	✓		
Recommendations, *Therapeutics and Devices Committee*. Medical Society of Virginia to support 1) American College of Physicians' position on drug substitution and 2) physician dispensing of prepackaged drugs for fee/charge when in best interests of patient.	✓		
PRACTICE GUIDELINES			
Resolution E: Formulation of Medical Practice Guidelines (*Robert P. Nirschl, MD*). Safeguards for AMA and specialty societies in developing medical practice guidelines, e.g., preliminary polling of membership, continuing review/amendment. Referred to Council.			
Resolution F: Medical Practice Guidelines and Malpractice (*Robert P. Nirschl, MD*). Medical Society of Virginia and AMA to support medical practice guidelines contingent on passage of federal malpractice legislative reform.			✗
PUBLIC HEALTH			
Resolution PP: Virginia Poison Network (*Virginia Chapter, American Academy of Pediatrics*). Medical Society of Virginia to take position in support of establishment of Virginia Poison Network.		✓✓	
STATE LEGISLATION			
Resolution L: Medicaid Reimbursement (*Virginia Academy of Family Physicians*). Medical Society of Virginia to endorse/support legislatively concept of utilizing Resource-Based Relative Value Scale for Medicaid.		✓✓	
Recommendation on Certificate of Need, *State Legislative Committee*. Medical Society of Virginia to recommend delay of CON deregulation. Referred to Council.			
Recommendations on Preferred Provider Contracts, *State Legislative Committee*. Medical Society of Virginia to reaffirm opposition to any change in law prohibiting a PPO from discriminating among health care providers or excluding any provider willing to meet terms/conditions.	✓		
Resolution R: Caller Identification Blocking (*Michael C. Trahos, MD*), Medical Society of Virginia to support all state/federal legislation requiring telephone companies to provide caller ID blocking on a cost-free basis.	✓		
YOUTH/SCHOOL HEALTH			
Resolution A: Physical Education/Fitness Training (*Robert P. Nirschl, MD*). Medical Society of Virginia to endorse activities promoting physical fitness in schools/elsewhere.		✓✓	
Resolution B: Routine Newborn Circumcision (*Alleghany-Bath County Medical Society*). Medical Society of Virginia to develop/make available guidelines for informed consent in newborn circumcision. Amended/referred to Child Health Committee.			
Resolution S: Allergic/Asthmatic Reactions at Schools (*Fredericksburg Area Medical Society*). Medical Society of Virginia to seek law requiring all Virginia schools to have action plans for dealing with acute allergic/asthmatic emergencies, including use of epinephrine injectors (e.g., EpiPen®) and inhalation bronchial dilators.		✓✓	

Resolution/Recommendation, Sponsor, Description	Adopted	Amended, Adopted	Rejected
Resolution Z: Medicaid Reimbursement for Child Health Services (*Virginia Chapter, American Academy of Pediatrics*). Medical Society of Virginia to support state budgetary increases to improve access to child health services by enhancing Medicaid reimbursement.		✓✓	
Resolution LL: Youth Access to Tobacco Products (*Hampton Medical Society*). Medical Society of Virginia to take position in support of state law restricting youth access to tobacco and refer position to Council for action.		✓✓	
Recommendation, *State Bar Liaison Committee*. Medical Society of Virginia and Virginia State Bar to form physician/attorney speaking teams to address youth groups on drug abuse.	✓		
Recommendation, *Sports Medicine Committee*. Medical Society of Virginia to urge Virginia High School League to consider using body fat percentage as criteria for weight class in high school wrestling.	✓		
Recommendations 1, 2, 3 and 4, *Child Health Committee*. Medical Society of Virginia to support concept of comprehensive health education programs, including education to reduce injuries from firearms; support well-baby care coverage in all health insurance policies sold in Virginia; and seek legislation mandating second measles or MMR vaccinations for children by age 12 and recommending this for young adults as appropriate.		✓✓	
Resolutions C and D: MMR Vaccine (*Virginia Academy of Family Physicians*). Medical Society of Virginia to urge the state to require second MMR vaccine for children 4-6 years.			✗

WINNERS

Winner Wallenborn

IN THE FIFTIES, **Dr. White Wallenborn** interned in mixed medicine. But then, settling down in Charlottesville to specialization in otolaryngology, volunteerism captured his versatility, and he began racking up a mix of air accidents and alums, rescue squads and Rotary, Boy Scouts and barbershop quartets. This is precisely the kind of extracurricular record the Physicians' Community Service Award was designed to honor, and at the 1990 annual meeting of the Medical Society of Virginia, Dr. Wallenborn was revealed as the 28th recipient. Presiding President William H. Barney introduced the winner at the Saturday night banquet with a this-is-your-life summary of his qualifications.

Born in North Carolina, White McKenzie Wallenborn took an MD at the University of Virginia School of Medicine and is now in group practice in Charlottesville. He is a clinical professor at UVa, on the governing staff of Martha Jefferson Hospital, and on the boards of the Virginia Council for the Deaf and the Virginia Hearing and Speech Foundation. For ten years Dr. Wallenborn was a squad surgeon and member of the board of the Charlottesville-Albemarle Rescue Squad, and he has served on the board of the Emergency Medical Services Council of the Thomas Jefferson Planning District. He has been president of the Albemarle County Medical Society, the American College of Surgeons, Virginia Chapter, the Virginia Society of Otolaryngology/ Head and Neck Surgery, and his alma mater's Medical Alumni Association, and a director of the Shenandoah PSRO.

In 1957 White Wallenborn graduated from the United States Air Force School of Aviation Medicine, thereby embarking on 25 years of USAF Reserve service as a flight surgeon. He is a member of the Civil Air Patrol, the Charlottesville-Albemarle Joint Airport Commission, and the Aero Club of Albemarle. For the Flying Physicians Association he has served on the board of directors. He also pays dues to the Aircraft Owners and Pilots Association. The Federal Aviation Administration relies on him as senior aviation medical examiner and aircraft accident investigator. (His extensive authorship includes an article on "Airport Medical Disaster Planning" in the April 1978 issue of *Airport Management Journal.*)

To this mix add standout performances with the Charlottesville United Way, Rotary Club, and Boy Scouts, and then sprinkle the seasoning: he's a camera-carrying member of the Virginia Society for the Photographic Arts, plays the bagpipe in the Albemarle Highlanders Pipe Band, and contributes a spirited baritone to the Charlottesville Chapter, Society for the Preservation and Encouragement of Barbershop Quartet Singing in America.

Concluding his encomium, Dr. Barney called Dr. Wallenborn to the dais and gave him a plaque. The

banqueters gave him a great big hand.

A pediatrician, **Dr. Gerald W. DeWitt**, Newport News, won first prize in the scientific exhibit section for his presentation of "The Effects of Maternal Intravenous Dextrose on Newborn Hypoglycemia." Second prize went to **Dr. William E. Kelley** and **Dr. Eric P. Melzig** of the Richmond Surgical Group for their exhibit on "Laser Laparoscopic Cholecystectomy." And **Dr. Kevin C. O'Loughlin**, of Eastern Virginia Medical School's Department of Plastic Surgery, took third prize with his exhibit on "Breast and Abdominal Body Contouring in the Morbidly Obese Patient Following Vertical Banded Gastroplasty."

These lucky members won prizes at the scientific exhibit booths: **Dr. Thomas M. Fulcher**, Falls Church, a kaleidoscope at the Haycraft Insurance booth; **Dr. Phillip B. Fuller**, Fredericksburg, the comforter raffled by Sleep Dynamics; from the National Safety Associates' display **Dr. Harry S. Holcomb III**, Franktown, took home an air purifier and **Dr. James L. Van Dyke**, Abingdon, a water purifier; Pearl & Associates' lady's and man's watches were won by **Dr. Thomas C. Iden**, Berryville, and **Dr. Jack C. Turner**, Danville; to **Dr. Duvahl B. Ridgway-Hull**, Roanoke, went the St. Albans Hospital's Cookbook; and "Heartcare" videos offered at the Virginia Cattle Industry Board's booth went to **Dr. George R. Smith, Jr.**, Shawsville, and **Dr. Joshua P. Sutherland**, Grundy.

The golf and tennis tourneys drew big turnouts. Emerging victorious as the new MSV golf champion was **Dr. Ellsworth J. "Russ" Stay** of Arlington, who carded a 75 low gross and took home the Challenge Cup. Second low gross was scored by **Dr. John C. Rhoades**, Manassas, and third by **Dr. Norman R. Edwards**, Newport News. In the low net division, a 75 was wrested from the Homestead course by **Dr. Earl E. Virts**, Purcellville, to give him first prize. Close behind in second was **Dr. William W. Regan**, Richmond, and in third, **Dr. Walter W. Schroeder**, Norfolk.

Dr. Byrd S. Leavell, Jr., Charlottesville, was the star of the tennis tourney, winning the men's singles. **Dr. William E. Kelley, Jr.**, Richmond, was the runnerup.

Dr. Hazle S. Konerding, Richmond (right), and Cynthia Boggs at the Virginia Cattle Industry Board's exhibit.

On the Homestead course: from left, Dr. Juan M. Montero II, Chesapeake; Dr. William W. Regan and Dr. Lawrence C. Zacharias, Richmond; and Dr. Albert L. Roper, Hampton.

VMQ
WINTER 1991

In Vitro Fertilization: Effective Alternative to Surgery for Distal Tubal Occlusion

Dimitrios K. Hassiakos, MD, Suheil J. Muasher, MD,
Lucinda L. Veeck, MLT, and Howard W. Jones, Jr., MD,
Norfolk, Virginia

A total of 512 consecutive patients with tubal infertility underwent ovarian hyperstimulation for the purpose of IVF-ET, resulting in a total of 829 attempts and 890 transfers (fresh and cryo). Overall, 218 clinical pregnancies were achieved, of which 114 (52.3%) resulted in live births, and 38 (17.4%) were ongoing (≥20 weeks of gestation) at the conclusion of the study. The total live birth and ongoing pregnancy rate per patient was 29.7% (three patients conceived twice). The probability for a patient to achieve a live or ongoing pregnancy was 42.7% after three attempts and 85.3% after six attempts. We conclude that IVF-ET using contemporary technology offers an effective alternative to surgery for tubal obstruction. Va Med Q 1990;118(1):26-30

TUBAL reconstructive surgery for the treatment of infertility secondary to pelvic inflammatory disease has been traditionally associated with rewarding but limited success rates.[1,2] The refinements in modern microsurgical techniques have added only modest improvement.[3-9] The current belief is that pregnancy rates following tubal surgery are more closely related to tubal damage and status than to surgical technique per se. Several workers have emphasized the importance of better patient selection for microsurgery and the preoperative identification of those with a poor prognosis who may benefit from in vitro fertilization and embryo transfer (IVF-ET)[1,3,10-15]

The worldwide expansion of IVF programs, the recent advances (i.e., use of GnRH analogues, transvaginal oocyte retrieval, cryopreservation of embryos) and the accumulating experience have led to a dramatic improvement in IVF pregnancy rates.[16-18] However, there are limited studies which have attempted to determine if current protocols of IVF-ET programs (which include added benefits from cryopreservation of excess pre-embryos) can offer patients with tubal infertility an equal or better alternative to tubal reconstructive surgery.[15,18-21]

The purpose of the current study is to assess the efficacy of IVF-ET after repeated IVF cycles, using contemporary methodology, in patients whose infertility was primarily attributed to a tubal factor. Further, our data will be compared to those following tubal surgery for distal tubal disease available from the existing literature.

From the Howard and Georgeanna Jones Institute for Reproductive Medicine, Department of Obstetrics and Gynecology, Eastern Virginia Medical School. Address correspondence to Dr. Jones at Hofheimer Hall, 6th Floor, 825 Fairfax Avenue, Norfolk VA 23507.

This text was presented to the Virginia General Assembly's Special Advisory Commission on Mandated Health Insurance Benefits for its public hearing on November 5, 1990.

Materials and Methods

A total of 512 consecutive patients whose primary diagnosis was tubal infertility and who underwent ovarian stimulation for the purpose of IVF-ET at the Norfolk program between January 1, 1987 and March 30, 1990, were retrospectively analyzed. The starting date was selected because cyropreservation became an integral part of the program at that time. The study

group included 829 consecutive attempts in 512 patients with a mean age (±SD) of 34.4 ± 3.8 years at the time of attempt. Of these, 416 (81.2%) were < 38 and 96 (18.8%) were ≥38 years of age. Ovarian stimulation protocols consisted of combinations of gonadotropins alone or with adjuvant GnRH analog (leuprolide acetrate, TAP Pharmaceutical, North Chicago IL) administered from the mid-luteal phase of the preceding IVF cycle by previously described protocols.[16,22] Laparoscopic or ultrasound guided oocyte retrieval was performed 32-36 hours after human chorionic gonadotropin (hCG) administration (10,000 IU intramuscularly). Oocyte assessment, sperm processing, fertilization procedures, cryopreservation, and embryo thawing and transfer were performed as previously described.[17,23,24]

For the purpose of analysis, patients were not categorized according to the degree and extent of pelvic disease or prior tubal surgery, as previously published data from our institute revealed no significant impact of these factors on the efficacy of IVF-ET.[19] Most patients had had unsuccessful tubal surgery. All cycles in which there was an attempted stimulation for egg retrieval were included, regardless of its success, in calculating the cumulative pregnancy rate per attempt. Further, preclinical abortions were not counted as pregnancies.

Statistical evaluation of the data included calculation of the observed cumulative pregnancy rates over time using the life-table method. For the evaluation of crude and cumulative pregnancy rate per attempt, fresh and cryo-derived pregnancies from any one harvest were considered part of a single attempt. When appropriate, comparisons between groups were made using the Fisher's exact test. All statistical calculations and modeling were performed using Statistical Analysis Systems (SAS).

Results

The study group consisted of 829 consecutive attempts from 512 patients with tubal infertility resulting in 890 embryo transfers (fresh + cryo). Only 50% of the frozen embryos had been thawed-transferred at the time of the study. Overall 218 clinical pregnancies were observed, with a pregnancy rate per patient of 42.6%. This yielded an overall pregnancy rate of 26.3% by attempt and 24.5% by transfer (24.3% for fresh and 25.4% for cryo transfers) as shown in Tables 1,2. If we consider fresh and cryo transfers per fresh transfers, then the calculated pregnancy rate is 28.7%.

Of the 218 pregnancies, 114 (52.3%) resulted in live births and 38 (17.4%) were ongoing (≥20 weeks of gestation) at the time of the study Twenty of the 114 pregnancies were multiple gestations (i.e., 15 sets of twins, 3 triplets, and 2 quadruplets). Of the 38 ongoing pregnancies, 12 had multiple gestations (11 sets of twins and 1 set of triplets). Also, 7 (3.2%) ectopic pregnancies were noted. The overall live birth and ongoing (≥wks) pregnancies rate per patient was 29.7% (3 of these patients conceived twice).

The cumulative pregnancy rates for all patients with tubal infertility as calculated by the life-table method are depicted in Table 1 and the corresponding curves are plotted in Figure 1. The expected probability of conception per attempt for fresh transfers was 49.7% after 3 and 88.0% after 6 attempts. The cumulative pregnancy rates per attempt for transfers with cryopreserved eggs plateaued after 3 attempts at 12.4% (Table 1, Fig. 1) because no patient had sufficient cryo eggs for more than 3 attempts. Taking into account both fresh and cryo transfers, the expected probability of conception increased and reached 57.4% and 89.9% after 3 and 6 attempts respectively. Further, a patient

Table 1. Life-table Analysis of Pregnancy Rates in All Patients.

	No. of IVF Attempts					
	1	2	3	4	5	6
No. of Attempts	512	189	81	30	14	3
Fresh Transfers						
No. of Pregnancies	123	40	13	5	2	2
Cumulative Pregnancy Rate/Attempt	24.0%	40.1%	49.7%	58.1%	64.1%	88.0%
Cryo Transfer						
No. of Pregnancies	20	10	3	–	–	–
Cumulative Pregnancy Rate/Attempt	3.9%	9.0%	12.4%	12.4%	12.4%	12.4%
Total (Fresh + Cryo)						
No. of Pregnancies	143	50	16	5	2	2
Cumulative Pregnancy Rate/Attempt	27.9%	47.0%	57.4%	64.5%	69.6%	89.9%
Live and Ongoing	101	33	11	3	2	2
Cumulative Pregnancy Rate/Attempt	19.7%	33.7%	42.7%	48.5%	55.8%	85.3%

*Ongoing: pregnancies ≥20 weeks gestation

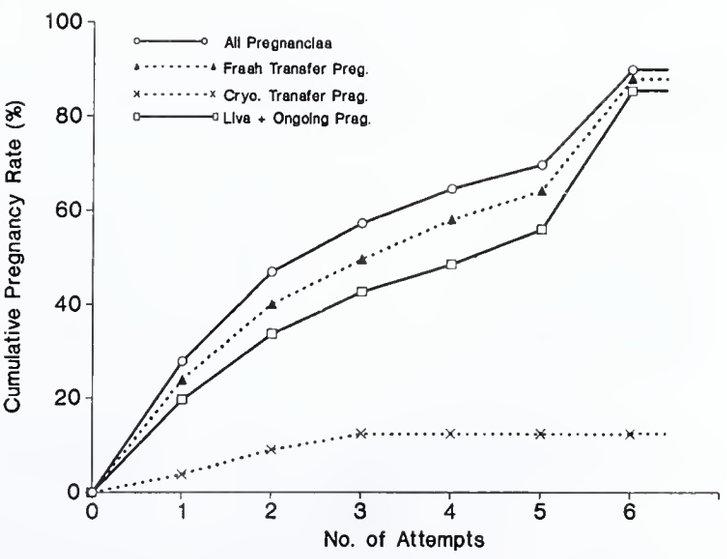

Fig. 1. Cumulative pregnancy rate (%) per attempt for all pregnancies (fresh + cyro transfer), live and ongoing (fresh + cyro transfer), and for pregnancies derived from fresh or cryo transfer alone.

can expect a 42.7% chance of achieving a live or ongoing pregnancy with 3 attempts and 85.3% chance within 6 attempts.

In analyzing our data with regard to the patient's age, a significantly higher overall pregnancy rate per attempt was observed in patients <38 years of age as compared to those ≥38 (28.3% versus 19.0%, $p < .02$). Similarly, a higher pregnancy rate (live birth and ongoing) per attempt was noted in the younger group compared to those ≥38 years of age (19.7% and 13.4%, respectively $p < .05$) (Table 2, Fig. 2).

Table 2. Life-table Analysis of Pregnancy Rates According to Age.

	No. of IVF Attempts					
	1	2	3	4	5	6
Patients < 38 years old						
No. of Attempts	416	151	58	17	7	1
Live and Ongoing	88	29	9	1	–	1
Cumulative Pregnancy Rate/Attempt	21.2%	36.3%	46.2%	49.3%	49.3%	100%
Patients ≥ 38 years old						
No. of Attempts	96	38	23	13	7	2
Live and Ongoing	13	4	2	2	2	1
Cumulative Pregnancy Rate/Attempt	13.5%	22.6%	29.4%	40.2%	57.3%	78.6%

*Ongoing: pregnancies ≥ 20 weeks of gestation

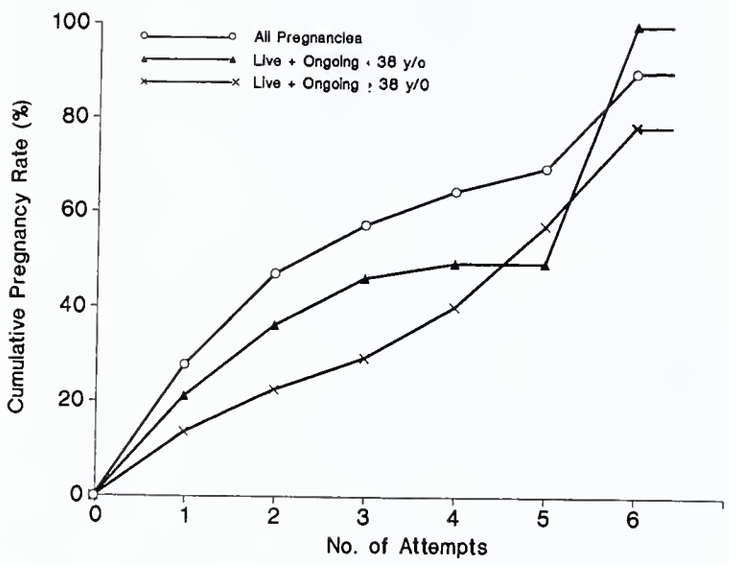

Fig. 2. Overall and live + ongoing cumulative pregnancy rate (%) per attempt according to the patient's age for fresh and cryo transfers combined.

Patients less than 38 years of age had a higher expected probability of conception (live birth and ongoing) after three and six attempts (46.2% and 100% respectively) versus those who were ≥38 years old (29.4% and 78.6%, respectively), ($p < .001$).

Expectation of Pregnancy after Tubal Surgery

In a recent review of the literature, Marana and Quagliarello[1] reported on the pregnancy outcome following tubal surgery for distal tubal occlusion. In these data obtained from 14 studies that included a total of 1275 patients with a 6-month to 8-year duration of followup, an overall pregnancy rate of 33% (range: 20-47%), intrauterine pregnancy rate of 26% (range: 12-47%), term pregnancy rate of 21% (range: 10-40%), abortion rate of 5% (range: 0-9%), and ectopic pregnancy rate of 8% (range: 0-18%) was noted.

However, information obtained from crude pregnancy rates in counseling patients regarding pregnancy outcome is limited by the variable length of followup present. Thus, in advising or counseling patients about their prognosis for a conception, cumulative pregnancy rates may be more helpful or reliable as length of followup is accounted for. Once such study that utilized life-table methodology to evaluate the pregnancy outcome for a number of microsurgical procedures for post-inflammatory tubal disease was reported by Jacobs et al.[25] In their study, the cumulative pregnancy rate and live birth rate for a median of 3-year follow-up among patients undergoing terminal salpingoneostomy was 47% and 32%, respectively. Also, the conception rate was greatest during the first two years postoperatively.

Similarly, in a recent review of the Johns Hopkins material on neosalpingostomy for distal tubal occlusion,[3] the highest cumulative pregnancy rate was observed among patients with mild disease, as categorized by the Rock classification, compared to those with moderate or severe tubal disease (50%, 35%, and 18%, respectively for the first operative year and reaching a maximum of 90%, 50%, and 25%, respectively for the following two years).

In examining the available data on cumulative pregnancy rates estimated by the life-table method following tubal surgery, the overall cumulative pregnancy and intrauterine pregnancy rates ranged from 4-38%, 10-45%, and 22-56% for the first, second, and third postoperative years, respectively.[3,12,13,25-27] The reported cumulative intrauterine pregnancy rates ranged from 9-20%, 14-27%, and 16-34%,[3,9] whereas the cumulative term or live pregnancy rates reached 18%, 26%, and 32%[25] for the first, second, and third postoperative years, respectively (Table 3).

Table 3. Cumulative Pregnancy Rates for Tubal Surgery for Distal Tubal Occlusion[3,9,12,13,25-27] and for IVF (Present Data).

	Tubal Surgery				IVF	
	Total#	IUP#	Live Births*		Total	Live Births + Ongoing
Postop Year				Attempt		
1st	4-38%	9-20%	18%	1st	27.9%	19.7%
2nd	10-45%	14-27%	26%	2nd	47.0%	33.3%
3rd	22-56%	16-34%	32%	3rd	57.4%	42.7%
4th	24-58%	19-34%	37%	4th	64.5%	48.5%

IUP = intrauterine pregnancy rate
# = range
* = average from all studies

There is little information in the literature concerning the pregnancy success rate for repeated salgingoneostomy and for distal and proximal obstructive tubal disease after tubal reconstructive surgery. In patients undergoing repeated salpingoneostomy, the reported intrauterine pregnancy rates ranged from 12-19% and

the term pregnancy rates from 6-16%.[2,11,26,28] Further, Lauritsen et al[26] reported a cumulative pregnancy rate of 10% for the first postoperative year and 18% for the following three years in these patients.

Similar findings have been observed in patients undergoing tubal surgery for distal and proximal occlusion of the oviduct. The intrauterine pregnancy rates reported ranged from 12-28%, with a term pregnancy rate of 0-28%.[2,11,14,25] A cumulative pregnancy rate of 21%-25% has also been reported after the second postoperative year for these patients.[14,25]

Discussion

The introduction of microsurgical techniques has rendered pregnancy outcome following tubal reconstructive surgery for postinflammatory disease only marginally better as compared with macrosurgery. However, it is difficult to compare results between studies due to the presence of a number of variables that affect the pregnancy success rate. Primarily, the differences in the classification systems used to categorize the extent of disease and the surgical technique, the lack of uniformity of patient selection, presentation of pregnancy data, and the lack of adequate followup makes comparisons and evaluations between studies difficult.[1,6,26-28] Nevertheless, it is obvious that despite atraumatic microsurgical tubal reconstructive surgery, most women do not achieve a live birth despite demonstrable high postoperative tubal patency.[5,9,25-26]

Current data support the belief that pregnancy outcome following tubal surgery is strongly related to the severity of preexisting tubal pathology and the extent of pelvic or adnexal ahesion formation.[3,10,28] Thus, the most severe form of the disease demonstrates a trend toward lower conception and live birth rates.

Various diagnostic factors based on the extent and severity of pre-existing tubo-ovarian disease have been described in order to more accurately predict the pregnancy success rate following tubal surgery and thus allow counseling of patients for the selection of optimal treatment modality.[10-15] It is clear that regardless of the classification system used to define the extent of tubo-ovarian disease, patients in the good prognostic class or category are those associated with mild disease. The chances of achieving an intrauterine or term pregnancy in this group of patients are excellent (60-77%). With patients in the intermediate or poor prognostic category, the success of achieving an intrauterine or term pregnancy are extremely poor (3-25%), and IVF-ET seems to offer a better alternative for these patients.[3,10,13] The available data on cumulative pregnancy rates expected in the first postoperative year range from 9-20% and reached a plateau at 19%-35% after the fourth year postoperatively.[3,5,13]

In the analysis of our data, IVF-ET in patients with tubal infertility were associated with an overall cumulative pregnancy and live-ongoing cumulative pregnancy rate of 57.4% and 42.7%, respectively after only three attempts, which increased up to 89.9% and 85.3% within six attempts, respectively. Thus, the tubal infertility patients enrolled in IVF-ET exceed within three attempts the highest cumulative live pregnancy rate (35%) observed after four years following tubal surgery regardless of age (Table 3). Further, in previously published data by our institute[19] it was demonstrated that neither the state of tubo-ovarian disease nor any history pelvic adhesions or previous tubal surgery had a significant impact on the efficiency of IVF-ET.

Therefore, better cumulative pregnancy rates were obtained within the first three IVF-ET attempts in patients with tubal infertility in our study population, regardless of the extent of tubo-ovarian disease present or patient's age compared to the highest rates reported in the literature for moderate-severe disease and comparable to those quoted for mild disease following tubal surgery. Also, as evidenced by our results, the risks of ectopic pregnancy and those of a major operation associated with tubal surgery are almost negligible in IVF-ET programs.[18]

Summary

In sum, the success rate of IVF-ET in patients with tubal infertility observed by our institute exceeds the expected live birth rate after tubal reconstruction. Given the poor probability of conception and the high incidence of ectopic pregnancy after tubal reconstructive surgery in patients with distal tubal occlusion, IVF-ET remains an effective alternative mode of therapy for patients with moderate and severe tubal disease and offers faster achievement of live birth, while avoiding the risk of major surgery.

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Primary Care and the Asymptomatic HIV Patient

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Virginia Medical Quarterly 1991;118(1):31-38

Primary Care and the Asymptomatic HIV Patient

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Most patients with HIV infections are asymptomatic or minimally symptomatic. Primary care physicians can play a unique and vital role in their care. They should: 1. Look for cases. Treatment can prevent or delay progression of disease. 2. Know how to interpret the serologic tests for HIV infection. 3. Collect an adequate data base to assess extent and severity of disease and screen for certain coexisting infections. 4. Intervene appropriately with counseling and prophylactic therapy. 5. Refer early when neoplasia, opportunistic infections, or diagnostic uncertainties occur.

Va Med Q 1990;118(1):31-38

PRIMARY CARE clinicians are exhorted to care for HIV-infected patients; duty and need are cited.[1,2] Many primary care clinicians hesitate; they prefer to refer to subspecialists or others with particular interest in HIV infection. Such referral is often appropriate and unavoidable, but the majority of patients with HIV infection are asymptomatic; if primary care clinicians don't diagnose and take care of them, no one will. This paper presents an approach to the asymptomatic or mildly symptomatic patient which is simple but thorough and which incorporates present standards of care. As in the case of any chronic disease, however, practice guidelines or rules of thumb must be applied with flexibility, taking into account the patient's desires and psychosocial situation as well as any co-morbid conditions.

See editorial comment on page 34.

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Submitted 8-12-90.

It should be noted at the outset that this paper is limited in scope and does not attempt to deal with some important issues, including patient reluctance to be tested, the roles of education and self-directed therapy, and the legal aspects of HIV infection and its treatment. Keeping these reservations in mind, the approach to the asymptomatic HIV-infected patient can be divided into five components: 1. Look for cases. 2. Make the diagnosis. 3. Collect data. 4. Intervene. 5. Refer.

1. Look for Cases

Why look for cases? Because for many asymptomatic patients intervention delays the onset of clinical illness. The attitude of yesteryear, that a positive test is a death sentence, the knowledge of which has no value, is passé. And yet many clinicians do not want to look for cases. Why not? The reasons are several.

They may fear getting infected themselves. The risk to primary care clinicians is real but can be reduced almost to the vanishing point by following protective protocols. Unlike their colleagues who use invasive procedures, the primary care clinician's exposure to bodily fluids is limited. A needlestick is probably the greatest danger. Studies by the CDC have shown that after a contaminated needlestick there is one chance in 250 that an individual will convert to HIV positive.

Wear gloves when doing venipunctures or handling any bodily fluids, and have the discipline not to hurry. Have bleach and gauze pads in the laboratory and each examination room to wipe away the drop of blood on the vacutainer stopper and to wipe up any spills. If your office laboratory is not automated, consider sending the blood to an outside laboratory.

Some clinicians avoid managing cases because they don't know how. Contrary to some published opinion,[1] primary care clinicians who see just an occasional HIV patient cannot know all about HIV infection and AIDS, but they can learn and apply the limited, accepted body of knowledge pertinent to the management of the asymptomatic individual. An outline of such management is presented here.

Another argument against looking for cases is that caring for infected individuals takes too much time. It does take time, but so does taking care of the patient with diabetes, chronic renal failure or any other multisystem disease. HIV infection is hardly special in this regard.

2. Make the Diagnosis

The diagnosis of HIV infection depends on serologic tests. But before testing, get informed consent. Consent need not be in writing (although many hospitals and practices do have patients sign consent forms), but the medical record should show that the patient consented—indeed, wanted—to be tested.

Some high-risk individuals who should be tested refuse. They may be afraid to find out that they are positive or they may fear that their job, health insurance or personal relationships may be jeopardized if they are known to be HIV-positive. These issues need to be realistically explored with the patient. The person who is today unable to cope with the possibility that he might have HIV infection may tomorrow request to be tested. But even subtle coercion may cause trouble.

Tell your patient that Virginia statute explicitly requires every physician treating any HIV patient to report the diagnosis and identity of the patient to the local health department. This is confidential information, used by the health department only for epidemiologic studies and contact tracing; there has been no recorded incidence of breach of confidentiality, but your patient should know that you are making the required report. Make sure that the patient knows, too, that the health insurance carrier will be aware that the test is being done unless the patient chooses to pay for the test out-of-pocket and not have the carrier billed. That will be fine if the test is negative, but if it is positive, the cost of subsequent management will probably require the use of insurance and so reveal the diagnosis. Some typical costs at the time this was written were: initial evaluation as described in this paper, $500-1000; zidovudine (AZT) at currently recommended doses, about $225/month; monthly aerosol pentamidine treatments for prophylaxis of PCP (pneumocystis pneumonia), at least $300; a 3-week course of fluconazole for *Candida* infection, over $500.

Discuss, too, the extent to which you can assure confidentiality. If you practice in an institution or large group, you may not be able to control who has access to the medical record; you should be open and honest about this before testing, to avoid trouble after testing.

If you and your patient decide to test, the initial test for HIV infection should be the ELISA (enzyme-linked immunosorbent assay). This test uses whole virus antigen preparations and so tests for antibodies against *any* HIV antigen. Hence, it has a very low false-negative rate but has a significant false-positive rate. The ELISA is reported as positive or negative. If the test is negative, no further testing is ordinarily done, unless the overall clinical picture warrants a high index of suspicion (see below).

If the ELISA is positive, a Western blot test should be done. The Western blot tests for antibodies to specific HIV antigens. The results should be reported as positive, negative or indeterminate.[3] If the test is reported in a confusing or noncommittal way, use another laboratory.

A positive test means that antibodies were detected against at least two of the three following HIV virus antigens: p24 (a group-specific protein antigen), gp41 (a glycoprotein envelope antigen), or gp120/160 (envelope glycoproteins 120 and 160 are often difficult to distinguish).[4] An indeterminate result means that antibodies against HIV virus were detected but did not meet the above criteria. A negative test means that no HIV virus antibody was detected.

Interpretation of the test results is fairly straightforward. If the ELISA is negative, the patient should be told that the test was negative. If there has been recent exposure or continued exposure to HIV infection, the test should be repeated at intervals dictated by the clinical circumstances.

If the ELISA is positive and the Western blot is positive, the patient has HIV infection, and should be managed accordingly.

If the ELISA is positive and the Western blot is negative, the patient should be considered to have a false-positive. Followup testing may be indicated, just as in the case of the patient with a negative ELISA.

If the ELISA is positive but the Western blot is indeterminate, the diagnosis is uncertain. Most, but not all of such individuals will be found not to have HIV infection[5,6] but until that is established with certainty they should be counselled about protecting themselves and their sex partners. The patient should be retested periodically (every six months has been suggested) and/or be tested for viral antigens (see below). They should assume they are infected until conclusively proven otherwise and take appropriate precautions.

Since both the ELISA and Western blot test for the

presence of antibody, they will not detect the infected person who has not yet had an antibody response. The duration of the interval between infection and the development of a positive antibody test is under active investigation. Although most persons will develop antibodies within three months of infection[7], some will take 18 months or even longer.[8] It is possible that these individuals, who are infected but seronegative, can be detected by testing for the presence of the virus p24 antigen or for the presence of viral DNA. While these tests are not yet suitable for routine clinical use, they might prove helpful in the patient with an indeterminate test or in individuals who test negative but have a very suggestive clinical picture.

3. Collect Data

First, conduct a thorough conventional medical anamnesis. If the patient has been a blood donor since 1985, when HIV testing began, this may date the seroconversion. A history of shingles or idiopathic thrombocytopenic purpura (ITP), both of which are common early in HIV infection, may also indicate how long the patient has been infected. Inquire about other common sexually transmitted diseases. A positive serologic test for syphilis may present a difficult problem because the usual treatment for syphilis may not be adequate in the HIV-infected patient,[9,10] and there is concern about relapse or activation of latent infection. The problem of syphilis is further complicated

COMMENTARY

HIV: A Primary Care Disease?

Dr. Link's article is both timely and appropriate. Timely because the number of individuals infected with the human immunodeficiency virus (HIV) continues to rise, and every segment of our population is now represented in this illness. In time, this implies that each and every physician will have occasion to minister to the HIV-infected person. With its myriad of presentations, manifestations, and complications, I cannot think of a specialty that will not have contact with these patients. As physicians it is incumbent upon all of us to learn about this disease in order to properly treat those entrusted to our care.

Dr. Link's suggested approach is appropriate both in its emphasis on the primary care provider and in its information. We know the time span from exposure to the actual development of the last stage of HIV infection (AIDS) is approximately ten years. The results of on-going research suggest that early intervention, before the development of AIDS, delays its appearance, thereby prolonging the quality and quantity of life. The patient will be under the aegis of the primary care provider for an even longer time before referral may become necessary. Dr. Link's recommendations for management are the current state of the art.

The theme of Dr. Link's writing encompasses the time only after a patient is known to be HIV positive. When should the physician suggest testing? A detailed lifestyle history should be part and parcel of the medical encounter. Sexual orientation, sexual activity, and sexual practices must be an area of inquiry. The use and abuse of chemical substances should be elicited. Not only intravenous drugs, but alcohol, marijuana, and cocaine (crack), as their use may lead to risky behaviors. All stereotypes of who falls into the above categories have crumbled as this has become a routine part of the history. Every human is subject to what are referred to as frailties, and the virus is not discriminatory. Lastly, one needs to learn of past transfusions and possible injections while visiting another country. This history taking should be routine, not only for detecting disease but also for discussing preventive measures.

At some time after exposure (usually 2-12 weeks) there is an acute infection presenting only as a "flu-like illness." How often have you considered HIV in this setting? Later, lymphadenopathy develops. Do you include HIV serology in the battery of tests ordered for such an evaluation? Still later in the course of HIV infection, but before the development of AIDS, varicella-zoster (shingles) may be seen. Do you routinely obtain an HIV test on all of your patients with zoster? In the workup and management of the woman with recurrent, chronic, recalcitrant *Candida* vaginitis, an HIV test should be obtained. All of the patients listed here present to the primary care provider.

As HIV infection is caused by a virus that was unknown until a few years ago, and leads to complications that once were so exotic as to be a cause for

because, rarely, some infected patients may have negative serologic responses. Find out if the patient has had hepatitis; enteric infections or infestations such as shigellosis or giardiasis; or oral lesions or skin lesions. Inquire about alcohol and other recreational drug use because these agents may impair immune responses, stimulate impulsive risky sexual behavior or, in the case of shared needles used for intravenous drug, allow direct transmission of infection.

The physical examination should include a baseline measurement of body weight. Fundoscopic findings should be recorded. The asymptomatic patient is most unlikely to have histoplasmosis of the retina, but it may be helpful to note preexisting drusen, exudates or other abnormalities. Generalized lymphadenopathy is common in the otherwise asymptomatic patient and is not necessarily a bad prognostic sign, but a change (waxing or waning) may indicate progression of disease. Scrutinize the skin. HIV infection is commonly associated with a variety of neoplastic lesions (especially Kaposi's sarcoma, which may be the first clinical manifestation of HIV infection) and nonneoplastic infectious and noninfectious skin diseases.[11] Look for evidence of peripheral neuropathy, although it is unlikely to be present in the asymptomatic patient. Finally, keep in mind that dementia may be the first symptom of HIV infection and try to assess the patient's mental status. An interview with a relative or companion may be helpful in this regard.

Blood tests should, of course include a complete blood count and differential, including a platelet count. Measure the total T4 (CD4+, helper/inducer) cell count (normal is usually $>500/mm^3$, but values may differ from laboratory to laboratory), the T4% (of total lymphocytes)—(normal >35), and the T4/T8 (helper/suppressor) ratio. The critically low values are the three 2's: 200, 20, and .2, respectively. These tests are a guide to both prognosis and therapy. If your laboratory offers an absolute T4 count without the other cell counts and ratios, this will supply almost as much information for considerably less cost. When dealing with a patient with AIDS, or a symptomatic HIV-infected patient without AIDS, many factors other than the cell counts must, of course, be considered. Even in the asymptomatic individual, however, the counts are a guide and only a guide. The trend shown by serial counts must be considered, as well as the patients personal and financial circumstances, and most importantly, the patient's own wishes. Some patients fear medication and want to avoid taking any if at all possible; some want to miss no therapeutic opportunity regardless of risk or cost. These considerations properly influence your recommendations.

Test for the presence of p24 antigen in the blood; p24 may be present shortly after infection before antibody tests (ELISA and/or Western blot) turn positive. When antibodies appear, p24 antigen usually becomes undetectable, only to reappear when the disease progresses. For unknown reason, the antigen may again become undetectable late in the course of the illness. The test must, therefore, be interpreted in the context of the entire clinical picture.

Many clinicians measure B_2 microglobulin levels (normal <3.0 mg/L) as an indicator of prognosis and extent of disease. Levels greater than five are associated with progression to serious disease within three years for 70% of untreated patients.[12]

The presence of hepatitis B surface antigen in the asymptomatic individual indicated the presence of a carrier state, and hepatitis B core antibody titer will indicate immunity or susceptibility, and so can be used to identify persons who might benefit from the hepa-

Grand Rounds, there is little experience on the most effective and optimal management of these patients. Well-designed clinical trials (i.e., science) are needed to answer the multitude of questions. The National Institutes of Health (NIH) originated the AIDS Clinical Trials Units (ACTU) to undertake these studies. These units are important and have provided much information, and will continue to be productive. But the NIH has acknowledged the primacy of the patient. It has done so by developing the Community Programs for Clinical Research on AIDS (CPCRA). The CPCRA is taking clinical trials to individuals who do not have access to an ACTU and/or who have been underrepresented in AIDS research. It is involving the primary care physician. Among the ever important science, the therapeutic potential of the a patient-doctor relationship has been recognized. If ever there was a disease that demands continuity of care and the healing inherent in a positive relationship, HIV infection is it.

Dr. Link is a member of the Richmond AIDS Consortium. This is one of the units funded by the NIH through the CPCRA. Through this mechanism, promising new therapies are able to be provided to the population of central Virginia. I encourage primary care providers to become involved.

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titis B vaccine.

Order a serologic test for syphilis (STS) with titers. If the STS is positive, the diagnosis should be confirmed with a treponemal test (such as the FTA-abs). An STS titer may be helpful to follow the response to any treatment, and will serve as a baseline if a relapse or reinfection is suspected.

Cytomegalovirus (CMV), toxoplasmosis and Epstein-Barr virus (EBV) antibody titers are of less certain utility. An initial high titer may indicate that the patient is at risk for reactivation of a latent infection. This information may become important if studies now underway discover effective prophylactic regimens. If the initial titers are low, a subsequent significant acute rise may confirm a clinical suspicion of active infection.

Do a tuberculin (PPD) skin test. The significance of a negative test will be greatly enhanced by simultaneous controls. If the controls are also negative, the patient may be anergic, in which case the negative PPD will be meaningless. Control skin test can be obtained by using standard, commercially available, cell-mediated immunity (CMI) tests (such as Multitest CMI—Merieux Institute, Inc., PO Box 523980, Miami, FL 33152-3980). If the patient is anergic, the skin tests are of no help and other diagnostic methods must be used.

Finally, order a chest x-ray. Pneumocystis pneumonia, mycobacterial pulmonary infections, interstitial fibrosis and "ordinary" pneumonias occur regularly. A chest x-ray may detect unsuspected disease, or, if negative, may later be useful as a baseline.

All this data should be gathered as soon as the diagnosis of HIV infection is established.

4. Intervene

Intervention can conveniently be categorized as follows: counselling; immunization; antiretroviral therapy; prophylactic therapy; and treatment of intercurrent problems.

Counsel. Counselling may help your patient cope with the illness and treatment. For the asymptomatic patient, counselling may be the most important intervention of all. You will need all the help you can get; many communities have groups that provide physical and emotional support for the "PWA" (person with AIDS). But you must deal with some issues yourself. The first, of course, is prognosis. Don't make light of the situation but be as optimistic as is reasonable. Ten years after seroconversion many HIV-infected persons are still asymptomatic. The prognosis for extending life for all patients with HIV infection is improving yearly and major advances in therapy are conceivable. Find out if the patient has shared the bad news with anyone; it is probably better if someone beside you and your patient knows. Make sure the patient understands his own infectivity and knows what safe sex is. Many patients, at least initially, stop having sex when the diagnosis is made, so there may be no need to dwell on that topic during the initial visits. Don't cover too much ground in one visit, but do give specific recommendations. No drugs, no alcohol, plenty of rest and exercise. Many patients become interested in diet and vitamins and there are many notions current, but no good data, so use common sense. It has been reported that sunlight stimulates the growth of HIV[13]; whether this is clinically important or not, using a sunscreen is probably a good idea. Most important, of course, is to listen, and to involve the patient in the making of the clinical decisions. As in any other chronic disease, the patient who is well informed and who is an active partner in treatment is likely to fare best.

Vaccinate. Most of the literature concerning infection in HIV-positive patients concerns opportunistic and unusual infections, but HIV patients are, of course, susceptible to "conventional" pathogens as well. Indeed, they may be more susceptible, and once infected, may have impaired immune responses. These patients should, therefore, be immunized against common pathogens. Late in the course of disease the antibody response to vaccines may be impaired or absent, so it is important to immunize as soon as possible.

All patients who are not already adequately immunized and who don't present a specific contraindication should receive polyvalent pneumococcal and *H influenzae* vaccines, diphtheria and tetanus toxoids (Td, adult form) and yearly influenza vaccine. If serologic tests show that the patient is not immune to hepatitis B, vaccination should be recommended if there is a possibility that the patient will be exposed to infection (usually sexual contact or intravenous drug use).

Other immunizations may be appropriate for the traveller or those with special occupational risks.

There has been some concern that stimulation of the immune system by vaccines may accelerate the HIV infection, but this is not known to have occurred. With the exception of live polio vaccine (and of course, the now obsolete smallpox vaccine) all the usual vaccines are considered safe for HIV patients. Polio immunization should be induced by the inactivated IM preparation.

Antiviral Therapy. Zidovudine, commonly referred to as AZT, is the only FDA-approved drug for use in treating HIV infection. Early in the history of its use, zidovudine frequently caused severe toxicity, but then it was being used in large (1200 mg/day) doses in very sick patients. The currently recommended dose, 500-600 mg/day divided into four or five doses, is usually well tolerated by the asymptomatic patient. Dosage, toxicity, and monitoring schedules are adequately described in the package insert.

Patients who cannot tolerate zidovudine (usually because of nausea, headache or bone marrow toxicity)

should be referred for possible inclusion in a protocol study. Interferon, 2′3′-dideoxyinosine (ddI) and other medications are currently being tested. If your community does not have a research center, you or your patients can obtain information concerning various AIDS trials by calling 1 (800) TRIALS-A.

If your asymptomatic HIV patient's T4 count is over 500, no specific antiviral therapy is indicated. Check T4 counts and other prognostic indicators at intervals of six weeks to six months, depending on how close the count is to 500 and if there is a downward trend.

If the T4 cell count is below 200, always prescribe antiviral therapy unless there is a compelling contraindication.

When the T4 count is between 500 and 200, there is some uncertainty as how to proceed.[14] There is data (some still unpublished but in the hands of the FDA) showing that, at least in the short run, therapy delays progression of disease.

These data, unfortunately, do not demonstrate improvement in survival or quality of life. Other concerns also exist. Some studies have shown that zidovudine loses it efficacy after 1½-2 years. This may not occur in patients with high T4 counts, but there is some concern that the zidovudine will become ineffective (through viral resistance or other mechanism) and not be useful when it is needed later in the course of the illness. Furthermore, many patients with high counts will remain asymptomatic for years and so may be needlessly exposed to the still unknown effects of long-term treatment.

Nonetheless, it is increasingly common practice to treat patients with T4 counts between 500 and 200. If you are in doubt about how to proceed, discuss the situation with your patient and take his desires seriously into account. Some patients are very reluctant to start therapy, others are eager. Also, repeat the count after a short interval, especially if it is near 500. A downward trend might indicate need for treatment, but a steady count in the high 400s might suggest that further close observation is in order. Take into account, too, the T4/T8 ratio, the T4/total lymphocyte count (T4%), the presence of p24 antigen, and the microglobulin level. These parameters may suggest that trouble is brewing and encourage use of zidovudine. An AIDS trial is also an option in these difficult situations.

Prophylaxis. Patient's whose T4 counts are below 200 are at particular risk for PCP and should be treated prophylactically. Initial studies showed that PCP could be prevented or postponed by administration of co-trimoxazole (trimethoprin/sulfamethoxazole) in relatively low doses. Commonly used regimens are one double strength tablet twice daily for 2-3 days per week or one tablet daily. Co-trimaoxazole is readily available, easily administered and inexpensive. Disadvantages are the additive (if not synergistic) bone marrow toxicity when used with zidovudine, and the inexplicable frequency, among HIV-infected patients, of rash, fever, or other adverse reactions.

Aerosolized pentamidine 300 mg/month is also effective and has a lower toxicity. It should be administered by aerosol devices, now readily available, capable of producing particles small enough (less than 4.0 microns) to reach the alveoli. Larger particles should be filtered out to avoid bronchospasm. Treatment may be administered through your hospital inhalation therapy department or a free-standing inhalation or home therapy service. PCP pneumonia may occur even with pentamidine prophylaxis. When it does, it is often atypical, occurring in the upper lobes, associated with cystic or cavitary changes; the patients are frequently much less ill than those PCP patients who have received no prophylaxis.

Patients with a positive PPD (and negative chest x-ray and no clinical evidence of tuberculosis) should be treated with INH if there is no pre-existing liver dysfunction. Liver function tests should, of course, be monitored.

Prophylactic treatments for other infections are under study, but none has yet been generally accepted.

Intercurrent Problems. Mucocutaneous diseases are common among HIV-infected patients. *H zoster* can be a serious problem. Treat these patients vigorously. If the face is affected or the lesions cover more than one dermatome or cross the midline, hospitalize the patient and treat with intravenous acyclovir. Treat those patients not requiring hospitalization with large doses of acyclovir (4000 mg/day: 4 capsules 5 times daily). Postherpetic neuralgia may be a serious problem, but corticosteroids should not be used because of their immunosuppressive effect.

Recurrent herpes simplex is also common in this group of patients. They can be treated with the usual doses of acyclovir (five 200mg capsules per day for five days). There is some hope that acyclovir has some antiretroviral effect, especially in combination with zidovudine. Many practitioners, therefore, continue low-dose acyclovir indefinitely after an eruption due to *H zoster* or *H simplex*.

Candida infection of the mouth is also common and suggests the presence of severe immunosuppression. The diagnosis is easily confirmed by a KOH preparation of oral scrapings. Topical therapy, such as clotrimazole troches, may be effective. But in severe cases or where dysphagia suggests the presence of *Candida* esophagitis, systemic therapy may be needed. Ketoconazole is effective but may cause liver damage. The newer drug fluconazole may be safer and equally effective but is extremely expensive. Other fungal skin infections may produce the whole spectrum of dermatophytoses. These too, may be difficult to eradicate with only topical therapy. Other infectious dermatoses include bacterial folliculitis, molluscum contagiosum and human papilloma virus (HPV) verrucae.

Common lesions of less certain etiology are sebor-

rheic dermatitis, alopecia, papular urticaria, and hairy leukoplakia (possibly caused by the Ebstein-Barr virus).

Otitis media and externa, sinusitis, pharyngitis and other respiratory infections should be tended to promptly. Because unusual organisms may be present, throat and sputum cultures should perhaps be done more often in HIV-infected patients than in the general population.

Pyogenic infections include paronychia (sometimes due to *Candida*), boils and furuncles and perirectal abscesses. Gonorrhea and chlamydia infections should be treated in the usual way, but be sure to get posttreatment cultures.

5. Refer

Early and appropriate referral is an important part of primary care for the HIV-infected patient. The consulting clinician should always be informed of the patient's HIV status. If you cannot rely on the patient to inform the consultant, get permission from the patient to do it yourself. The consultant must know so that he can make an accurate clinical appraisal, and communicate effectively with you.

Patients with cough, fever, hypoxemia or pulmonary infiltrates may have infection caused by protozoa (pneumocystis or toxoplasmosis), mycobacteria (usually *M tuberculosis* or *M avium intracellulare*—MAI), fungi, mycoplasmas or common bacterial pathogens. The diagnosis cannot await the full-blown typical clinical picture because it may never appear (especially in patients who have been receiving prophylactic therapy or who have been partially treated) or may appear as a late manifestation. Prompt diagnosis requires early referral for bronchoscopy, bronchial brushings and washings and/or biopsy.

The patient with diarrhea should have routine stool cultures, examination for ova and parasites, and acid-fast-stain in search of cryptosporidium. Stool and urine can be cultured for CMV virus. If all these tests are unrevealing, a short course of a broad-spectrum antibiotic such as ciprofloxacin might be reasonable. If diarrhea continues, referral for consultation and/or endoscopy is appropriate.

New, severe or prolonged headaches may indicate CNS infection or neoplasm and should be investigated accordingly, as should, of course, dementia or focal neurologic signs. Neurologic disease can progress rapidly and should occasion early referral.

Any visual complaints should be taken seriously, and even asymptomatic retinal lesions should prompt referral to an ophthalmologist.

Skin lesions that you do not recognize, that may be neoplastic or that do not regress in response to your initial therapy should trigger referral to a dermatologist. Syphilis may present as a primary or secondary skin lesion, fever, adenopathy or CNS lesion. As we have already noted, treatment may be difficult, has to be individualized and will often require consultation with infectious disease experts.

Weight loss, unexplained fever, abdominal pain, liver dysfunction, anemia and other cytopenias and a host of other clinical manifestations may defy your diagnostic efforts and require referral.

Conclusion

Primary care physicians can play a vital role in the care and counseling of asymptomatic HIV-infected patients. The author provides a five-step approach to the diagnosis and management of these patients, with guidelines for interpreting tests, screening for coexisting problems, and prescribing prophylactic therapy.

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Clinicopathology Conference: Chronic Fever and Lymphadenopathy

From the Department of Medicine
University of Virginia School of Medicine
Discussed by John T. Philbrick, MD

Presentation of Case

Dr. Coleen A. McNamara: The patient is a 62-year-old white male who presented for evaluation of six weeks of fever, fatigue, anorexia, and cough.

He was previously an active, healthy man practicing as an anesthesiologist at one of Virginia's community hospitals. He had gone on a ten-day trip two months prior to presentation to Las Vegas, Nevada, and Tucson, Arizona. Most of his time was spent indoors, but he did visit a desert museum. He also made note of the fact that the first hotel he stayed in was very dirty, with significant mold and mildew on the shower curtain. After arriving home he felt as if he never caught up with his rest. One week after arriving home, while having dinner with friends, he felt suddenly tired and had no appetite. Two days later he began spiking temperatures to 102°F. Over the next six weeks he continued with intermittent fevers to 104°F with fatigue and anorexia. He also developed a dry, brassy, nonproductive cough. As his temperature would go up, his cough would increase. He noted some arthralgias in his knees and ankles. However, he also noted that this had been common over the years. During this time he was intermittently able to work because between fevers he felt well. He experienced a 7-8 pound weight loss over the six-week period. His initial evaluation by his local doctor included a negative ANA, RF, and monospot. Fungal serologies were negative. PPD was negative and fungal skin tests revealed a question of a slight reaction to histoplasmin. Chest x-ray revealed old calcified nodes with no acute process seen in the heart or lungs. Abdominal computerized tomography (CT) revealed a liver cyst and shotty retroperitoneal adenopathy. Tularemia and brucella titres were less than 1:80. At this point he was referred to the University of Virginia for further evaluation.

He denied visual changes, jaw claudication, muscle pain, pleurisy, photosensitivity, skin rash, shortness of breath, abdominal pain, chest pain, and changes in bowel habits. His past medical history included a myocardial infarction in 1980 treated with enteric-coated aspirin, and cervical degenerative joint disease. No history of transfusions. A history of needle stick exposure while working in an operating room six months previously; the patient was a child undergoing an ear operation who is doing well. No history of pets, no exposure to farm animals, to tuberculosis, to insects. Patient does not smoke, drinks two martinis a day usually, but is drinking less of late due to his illness. Denied IV drug use or any sexual risk factors for HIV.

Physical exam. T 99.3°F, BP 130/70, HR 82, RR 16. In general he was a thin white male in no distress. HEENT exam was unremarkable. His throat was clear, mouth revealed moist mucous membranes with no lesions, neck was supple without adenopathy or thyromegaly. There was no other adenopathy. The chest was clear to auscultation and percussion, and the

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heart showed regular rate and rhythm with no murmur, rub or gallop. His abdomen revealed active bowel sounds and was nontender with no masses or organomegaly. There was no joint redness, swelling or tenderness. Rectal exam revealed good tone, a large smooth prostate, and heme negative stool. Neurologic exam was nonfocal.

Laboratory data. WBC 5.1K with 66% PMNs, 12% lymph, 14% monos, 0.3% eos, 0.4% basos. Hematocrit was 33% with an MCV of 81 and MCH of 27. Platelets were 217K. Na 135mEq/L, K 4.7mEq/L, Cl 95mEq/L, CO2 23mEq/L, BUN 15mg/dL, creatinine 0.8mg/dL, calcium 8.5mg/dL, phos 3.6mg/dL, total protein 6.5g/dL, albumin 3.4g/dL, alk phos 70U/L, ALT 14U/L, AST 19U/L, CK 118U/L, lactate dehydrogenase (LDH) 541U/L. Arterial blood gas revealed pH 7.5, Pco_2 31, Po_2 100 on room air. Sedimentation rate 105mm/hr, reticulocyte count 1.0%. Hepatitis screen was negative, antiHIV antibody negative, RPR nonreactive. SPEP revealed increased alpha 2 globulins, a relative increase in alpha 1 and beta globulins and a relative decrease in albumin. Chest x-ray showed no acute process in the heart or lungs. He had mild degenerative changes in the spine.

Clinical course. The patient was seen in the infectious disease clinic. At that time his fevers were only low grade (99°F) and his appetite had picked up. Physical exam was unremarkable. Since he was improving, a more aggressive workup was deferred and he was referred back to his local medical doctor for followup fungal serologies. He did well until one week later when he developed a "head cold" and his fevers, fatigue and anorexia returned. He developed a productive cough and was treated with cefuroxime for increased sputum production.

He returned to ID clinic three weeks later with fever, chills, anorexia, fatigue, and a dry cough. Physical exam was unchanged except for two 0.5 cm firm, mobile, left supraclavicular lymph nodes. They were felt by the cytopathologist to be difficult to palpate, and needle aspiration revealed subcutaneous fat without lymphoid tissue. The patient was seen by pulmonary consultants, who felt he was unlikely to have parenchymal pulmonary disease as his chest x-ray was clear. He was admitted to the hospital for further workup of his symptoms. He continued to be febrile while in the hospital to 103.7°F. Blood cultures were negative, urine culture was negative, PPD was negative and the patient was anergic. Fungal serology for histoplasmosis, blastomycosis, and coccidioidomycosis were all <1:8. The patient was seen by ophthalmology who found cytoid bodies on exam. The patient continued to remain febrile but was otherwise stable. A diagnostic procedure was performed.

Radiology

Dr. Christopher L. Sistrom: Let's deal with the chest radiograph obtained on admission to this hospital first. The only findings were a small, calcified, solitary pulmonary nodule (a granuloma) in the right lower lobe and calcified subcarinal lymph nodes (Figs. 1A and 1B). Conditions which commonly produce

Fig. 1A. Frontal chest radiograph shows small granuloma (white arrowhead).

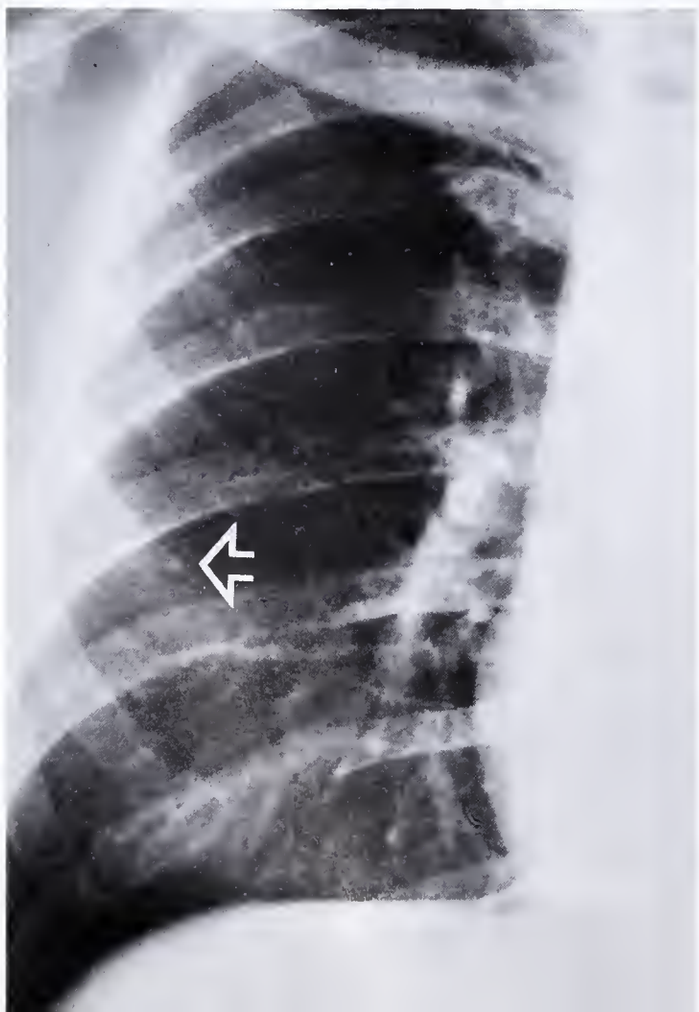
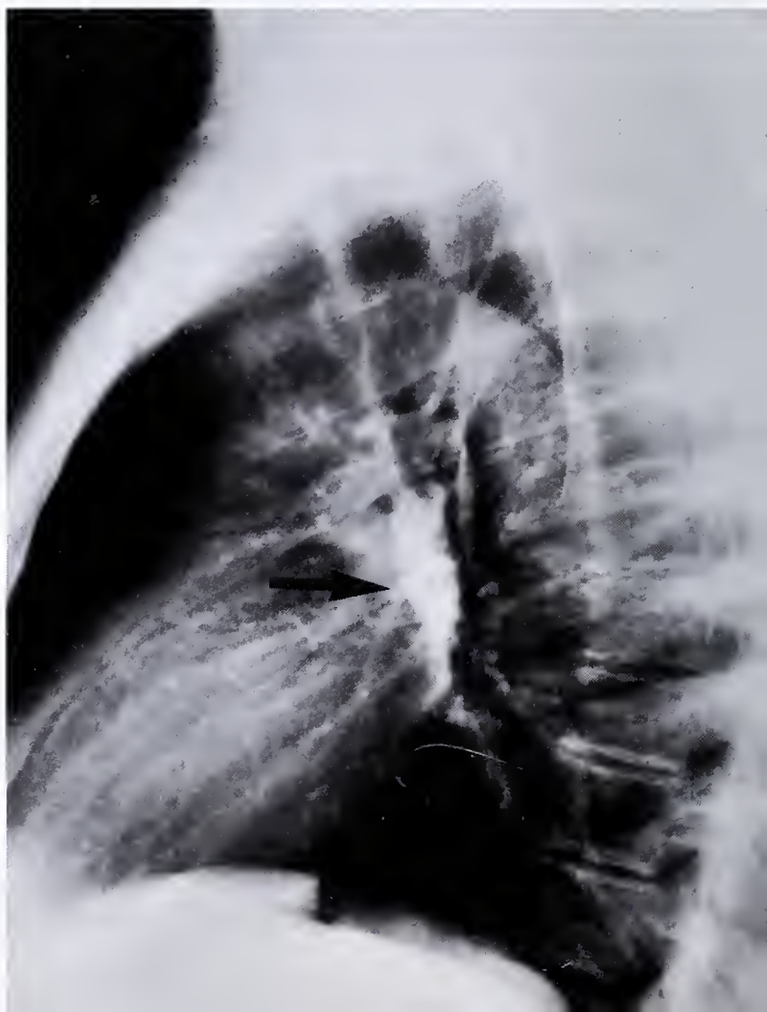


Fig. 1B. Lateral view depicts mat of calcified subcarinal lymph nodes (black arrow).



both of these findings are histoplasmosis, tuberculosis, and other fungal infections including coccidioidomycosis. However, granulomas and calcified lymph nodes are typically the sequelae of subclinical primary infection and take at least three months to manifest. Acute infection with coccidioidomycosis causes central nodal enlargement in one-quarter of patients, pleural effusion in about one-quarter, and patchy infiltrates in up to one-third, with resolution of these findings within a month. So I think that the chest x-ray findings are from previous histoplasmosis infection and do not relate to the problem at hand.

What are the chances that there are abnormalities in the patient's chest that are not seen on this chest radiograph? Recent comparisons of CT scanning of the chest and plain chest x-rays have shown that up to 50% more abnormal nodes can be found in the hila and mediastinum with CT scanning. Also, CT scanning will find almost twice as many small (that is less than 5 mm) pulmonary nodules as chest x-ray alone. Particular problem areas are the subcarinal, pretracheal, and anterior and superior mediastinal groups.

The liver finding (Fig. 2) is a cyst by strict CT criteria. It is solitary, it is small (about 1 cm), its CT density is that of water, and it has a spherical shape with regular walls. The liver is otherwise unremarkable. The only other abnormality of note in the abdomen are three enlarged lymph nodes in the para-aortic area at the level of the lower pole of the left kidney. The largest of these is 1.8 cm in diameter (Fig. 3). Usually we can judge whether or not lymph nodes are pathologic only by their size on CT scanning. Abnormal nodes rarely show necrotic centers and contrast enhancement. In the abdomen lymph nodes are considered normal if they are less than 1 cm in greatest dimension. That is not to say that small nodes do not ever contain pathology, but a large amount of experience has shown that the vast majority of small nodes are normal. Conversely, if a lymph node in the abdomen or pelvis is greater than 1.5 cm it is considered unequivocally abnormal.

The common causes of retroperitoneal lymphadenopathy are metastatic cancer, lymphoma, infections (tuberculosis is the prototype), and the lymph node syndrome of HIV. Of the lymphomas, non-Hodgkin's varieties are more likely to have abdominal involvement and the involved nodes are usually enlarged. Less than half of patients with Hodgkin's lymphoma have abdominal disease and only a third of those will show enlargement on CT scanning.

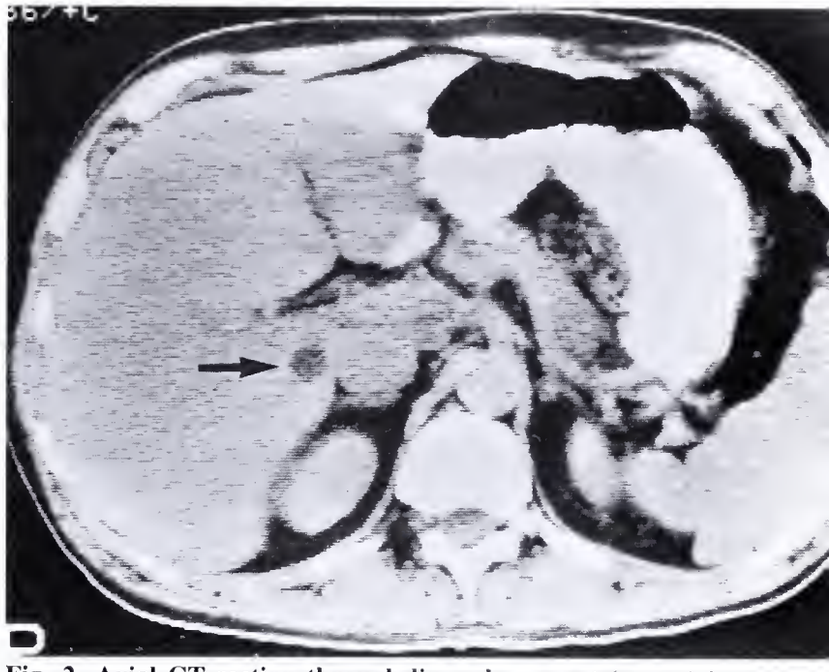


Fig. 2. Axial CT section through liver shows cyst (arrow) in right lobe lateral to IVC.

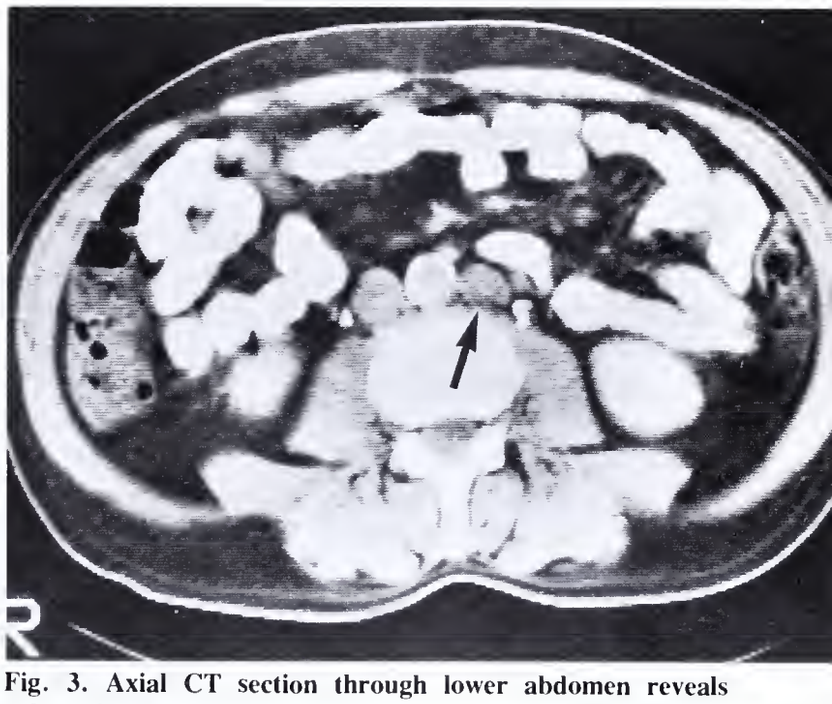


Fig. 3. Axial CT section through lower abdomen reveals enlarged para-aortic lymph node (arrow).

Clinical Discussion

Dr. Philbrick: Two assumptions guide my discussion of the case. First, because the course of the disease was prolonged, I shall assume that the symptoms and signs are fully developed and therefore likely to present a usual presentation of the disease rather than an uncommon one. Second, because the signs and symptoms do not significantly change in character over the course of the disease, I shall assume that only one disease process will account for this illness.

The major features of this disease, the fever and cough for 12 weeks, occurred in the setting of a previously healthy nonsmoker. The fever was moderately high with periods of remission of up to a week or more. The patient was ill but not toxic. In addition, the dry cough, normal lung exam, normal lung fields on chest x-ray, and normal arterial blood gases support the conclusion that the patient does not have a disease of the lung parenchyma. I therefore exclude from consideration chronic bronchitis, bronchiectasis, chronic obstructive pulmonary disease, asthma, pneumonias of all types, lung abscess, right middle lobe syndrome, and pneumoconioses. Mechanisms remain-

ing to account for the cough are endobronchial inflammation from infection or tumor, mediastinal nodes or mass not seen on chest x-ray pressing on the bronchi or nerves in the chest, or an abdominal process causing diaphragmatic irritation.

The lymph nodes were described as shotty, firm, and mobile. Although it is common to have a few cervical lymph nodes in healthy people, supraclavicular nodes are unusual and in this setting must be considered abnormal. The computed tomography scan of the abdomen implicates a second site of lymphadenopathy. The lymph node involvement is increasing over time, suggesting that the disease is slowly progressive. Despite an extensive workup no obvious primary malignancy to cause widespread adenopathy is evident. These observations rule out local infections and cancers unlikely to be both occult and associated with lymph node metastases in multiple sites.

Other features of the patient's history, physical, and laboratory evaluation that must be taken into account are the travel history that raises the possibility of exposure to coccidioidomycosis; the chest x-ray calcifications, which are typical of histoplasmosis; anergy, which can be caused by many different infections and tumors; the anemia, a normocytic, normochromic type with a low reticulocyte count, a pattern common for anemia of chronic disease; the lymphocytopenia; and the extreme elevation of the erythrocyte sedimentation rate, seen most often in malignancy, myeloma, infection, and collagen vascular disease. The slightly increased LDH without other liver function abnormalities suggest hemolysis or tumor. The increased globulins on the serum protein electrophoresis is a nonspecific reaction to many diseases including infection and tumor. Of note, there is no monoclonal spike. The history of a needle stick raises the question of hepatitis or HIV risk.

My initial differential diagnosis list (Table 1) focuses on conditions that can cause prolonged fever and lymphadenopathy.

Epstein-Barr virus (EB) and cytomegalovirus (CMV) are the two most common causes of the mononucleosis syndrome, so called because of the characteristic hematologic picture of greater than 50% mononuclear leukocytes and greater than 10% atypical lymphs. EB virus is the usual cause of the infectious mononucleosis syndrome that occurs in adolescents and young adults and is characterized by cervical lymphadenopathy, fever, and sore throat. Splenomegaly occurs in about half, hepatitis in a smaller fraction. The heterophile and monospot tests are usually positive. The illness lasts 4 to 8 weeks. In older patients, EB virus infection may be less typical, presenting as pneumonitis, CNS infection, mono or polyneuritis, myocarditis or pericarditis, hemolytic anemia, thrombocytopenia or agranulocytosis. CMV mononucleosis is quite different, with lymph nodes and pharyngitis occurring in few patients. Hepatitis occurs in more

Table 1. Initial Differential Diagnosis.

Viral diseases	Deep fungal infections
Infectious mononucleosis	Histoplasmosis
Epstein-Barr virus	Coccidioidomycosis
Cytomegalovirus	Sarcoidosis
Human immunodeficiency virus	Tuberculosis
Protozoa	Tumors that cause fever
Toxoplasmosis	Lymphoma

than half of patients with CMV infection. The illness usually lasts 2 to 6 weeks. CMV infection can occur in the immunocompromised host, often causing pneumonia, hepatitis, retinitis, or colitis. I exclude these two mono syndromes as a cause of disease in this patient because the clinical course of the patient does not fit. The mono spot test is negative, cough is unusual for both diseases and the peripheral blood smear on several occasions did not show the characteristic changes.

Primary HIV infection recently has been recognized as another mononucleosis syndrome. The incubation period is 2 to 4 weeks after infection with the virus. Although the illness is often subclinical, when recognized it is characterized by fever, pharyngitis, and lymphadenopathy that is usually axillary, occipital, or generalized. The lymphadenopathy is most common in the second week of illness before decreasing. Other symptoms that may occur are headache, anorexia, nausea, vomiting, rash, and diarrhea. Early on, there is a transient decrease in lymphocytes, while later lymphocytosis and atypical lymphs may occur. HIV antibodies appear in 4 to 12 weeks.[1-3] Another HIV syndrome to consider is CDC III HIV infection, formerly known as AIDS-related complex, and this patient appears to fulfill the criteria for its diagnosis[4], but I am going to exclude both HIV-related syndromes because the patient denies high-risk behaviors, the remote needle stick injury appears to be benign, the lymphadenopathy is not impressive enough, and the HIV antibody test is negative.

Toxoplasmosis is usually acquired from ingestion of infected rare meat or cat excreta. Although the disease is often asymptomatic, when clinically apparent it is characterized by lymphadenopathy, typically symmetric in the anterior and posterior cervical areas but sometimes localized or unilateral. The nodes are rubbery and nontender. Fever occurs in about half the patients and is usually low grade, although it can be occasionally high and prolonged. Notably absent is cough as a symptom. The hematologic picture frequently shows a slight lymphocytosis. The illness lasts weeks and sometimes months. In the immunocompromised host, disseminated toxoplasmosis can occur and is usually fulminant and rapidly fatal.[5] I shall rule out toxoplasmosis because the lymph node pattern is atypical and the supraclavicular nodes developed too late in the disease course.

At first look the possibility of this patient having histoplasmosis or coccidioidomycosis seems high. The

patient's chest x-ray and his slight reaction to histoplasmin skin tests is strong evidence for prior infection with histoplasma capsulatum. The patient's trip to the southwest United States several weeks before his becoming ill makes coccidioidomycosis a possibility. Histoplasmosis is endemic in the Mississippi, Missouri, and Ohio River valleys, an area that includes western Virginia. Coccidioidomycosis is endemic in the southwestern United States. Transmission to humans is effected by inhalation of the fungus with an incubation period of 1 to 3 weeks. For both diseases, the primary infection is usually asymptomatic. However, when symptomatic, they are characterized by fever, cough, and an abnormal chest x-ray in most cases. This patient had several chest x-rays that were negative; therefore, if he had histoplasmosis or coccidioidomycosis, the infection would necessarily have disseminated to an extrapulmonary site. Other than the lung, the sites of dissemination in these two diseases are lymph nodes, meninges, bones, joints, skin, liver, kidney. and adrenals. Dissemination is more likely to occur in patients who are immunocompromised, elderly, diabetic, chronically ill, or who are exposed to a heavy innoculum of fungus. Skin tests for both diseases are quite useful, but often are negative in disseminated disease. Serologic tests are usually positive seven weeks after infection, but may be negative in dissemination.[6] I conclude that this illness is not histoplasmosis or coccidioidomycosis because the patient's disease is not in the lungs, where it usually is for both infections, the patient has no risk factors for developing dissemination, the serologies are negative and there is no obvious site of dissemination other than the relatively mild lymphadenopathy.

Sarcoidosis is a disease of unknown etiology that causes fever in a significant minority of cases.[7] Although it is a disease of the young, about one-third of the cases occur in patients over age 40. About 90% of the time, it presents with intrathoracic disease, either prominent adenopathy and/or lung involvement. Peripheral lymphadenopathy is present in half the patients, but the nodes are often modest in size and unnoticed by the patient. The laboratory exam often shows lymphopenia and may reveal abnormal liver function tests. The serum electrophoresis may show increased globulins and anergy is frequently present.[8] In sum, sarcoidosis appears to be a real possibility in this patient since the age and fever curve are not unusual for sarcoidosis, a third of patients with fever and sarcoid have a relapsing fever course, and 10% of sarcoid patients with fever present with normal chest x-rays.

Tuberculosis (TB) always seems to be a consideration in clinicopathologic conferences. Although pulmonary TB can be ruled out because of the normal chest x-rays, miliary TB presents with fever and often with cough and lymphadenopathy. Anemia is occasionally found and the classic miliary pattern on chest x-rays occurs only 60-80% of the time. The tuberculin skin test is negative in about half the patients and anergy is often present. Although this patient has no history of positive PPD or risk factors for dissemination, TB is one of the great imitator diseases and a common cause of fever of unknown origin and therefore remains a consideration.

Finally, although myxomas, hypernephroma, osteogenic sarcoma, hepatic tumors and lymphoma all are known to cause prolonged fevers, in this case lymphoma is the only likely candidate. Lymphoma usually presents with lymphadenopathy and with fever in a quarter to one-half of patients. Lymphoma frequently may not involve the chest. Anemia, lymphopenia, elevated sedimentation rate, and anergy are often present.

My list of possibilities of disease in this patient includes sarcoidosis, miliary tuberculosis, and lymphoma, and I turn to the eye findings reported by the ophthalmology consultant. The cytoid bodies found, also known as "cotton wool" spots, are microinfarcts from arteriolar occlusion, often found with hypertension, diabetes mellitus, endocarditis, systemic lupus erythematosus, acquired immunodeficiency syndrome, and acute leukemia; but they are not reported in the conditions that I am considering.[9] However, I believe that the consultation was performed in an effort to discover evidence of sarcoid involvement of the eye. These are iridocyclitis, choroidoretinitis, and conjunctival and lacrimal gland involvement, occurring in around 25% of patients with sarcoidosis. I find it significant that these findings were not present.

I am going to follow my first assumption, which is that this disease is following a usual course rather than an unusual one, and since miliary TB and sarcoidosis typically present with chest findings, I will choose lymphoma as the likely diagnosis. Further, it is possible to come to some conclusion about what type of lymphoma it may be. Compared to non-Hodgkin's lymphoma, Hodgkin's disease is more likely to present with constitutional symptoms and have a "centripital" distribution of lymph nodes. Lymph nodes are more likely to involve contiguous areas in Hodgkin's disease. Abdominal involvement, although frequent in non-Hodgkin's lymphoma, is also common in older patients with Hodgkin's, especially those with fever and night sweats. Mediastinal nodes are present in about half of Hodgkin's disease patients, but only around 20% of those with non-Hodgkin's lymphoma.[10] I believe that the patient's clinical picture fits Hodgkin's disease best. The patient's fever could well be the Pel-Ebstein type, a cyclic fever with several days temperature elevation alternating with afebrile periods lasting for days or even weeks. Anemia, lymphocytopenia, anergy, markedly elevated sedimentation rate, increased globulins, and increased LDH are common in Hodgkin's disease. The cough could be from mediastinal nodes not visible on the

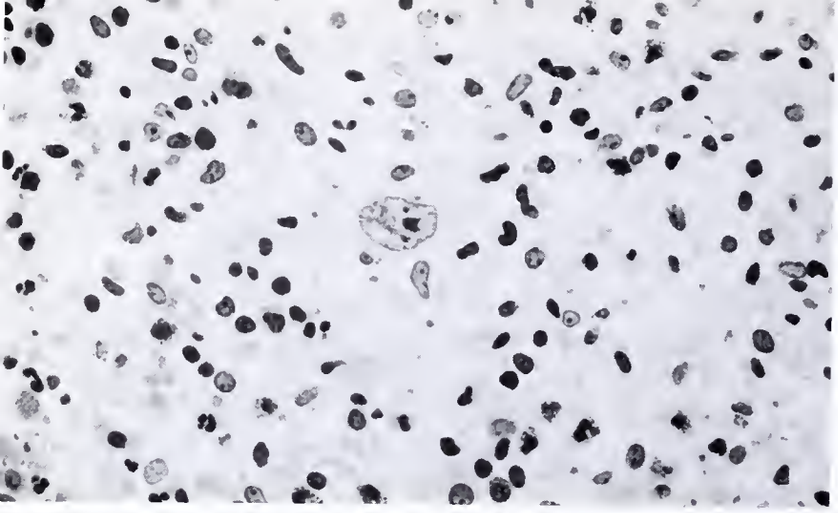


Fig. 4. Bone marrow showing fibrosis and pleomorphic cellular infiltrate of lymphocytes, histiocytes, plasma cells, eosinophils, and mononuclear Reed-Sternberg cell. H&E × 320

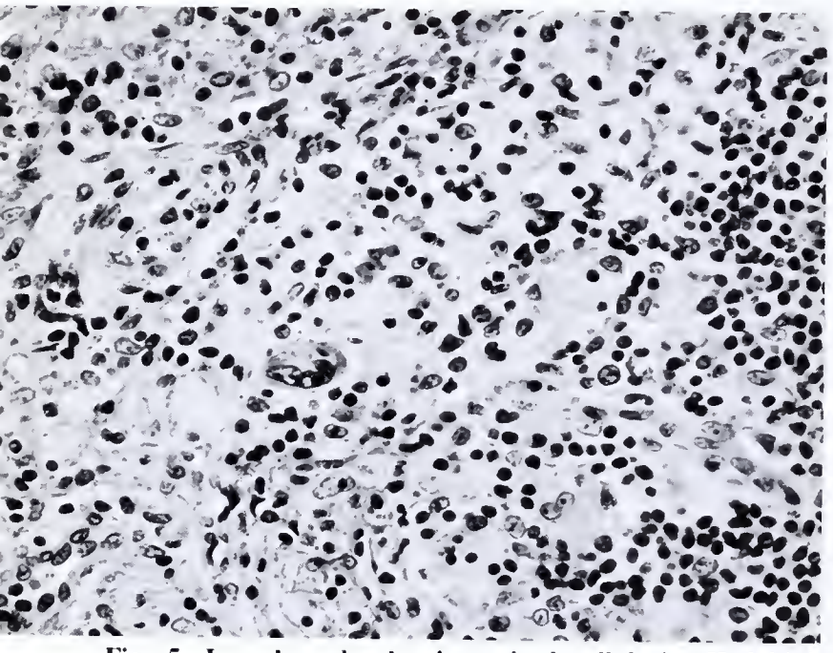


Fig. 5. Lymph node showing mixed cellularity Hodgkin's disease with multinucleated Reed-Sternberg cell. H&E × 320

chest x-ray or from abdominal involvement affecting the diaphragm. I choose a type of Hodgkin's disease that is common in older patients, often presenting with fever, weight loss and relatively rapid progression. This type of Hodgkin's disease often has left supraclavicular node involvement associated with retroperitoneal lymph nodes, with mediastinal involvement missing about 15% of the time. This disease is the lymphoctye-depleted type of Hodgkin's disease. The procedure is most likely an open lymph node biopsy of the supraclavicular nodes.

Diagnosis and Pathology

Dr. Benjamin C. Sturgill: The diagnostic procedure was a bone marrow biopsy from the posterior iliac crest. Most of the marrow was normocellular, but some areas were fibrotic with a pleomorphic cellular infiltrate consisting of lymphocytes, histiocytes, plasma cells, eosinophils and scattered larger cells, with vesicular nuclei and prominent nucleoli (Fig. 4). A diagnosis of Hodgkin's disease was made. Biopsy of a left cervical lymph node (Fig. 5) showed architectural disarray and a pleomorphic infiltrate of histiocytes, plasma cells, eosinophils and scattered Reed-Sternberg cells. A diagnosis of mixed cellularity Hodgkin's disease was made.

Dr. Daniel N. Mohler, Jr.: Could we have some followup on this patient?

Dr. McNamara: The patient was diagnosed as having Stage IV Hodgkin's disease. He received chemotherapy at a hospital close to his home. He improved markedly, with resolution of fevers and sweats after his first treatment. He gradually regained his appetite and strength such that he was able to return to work. Several months later he had recurrent malaise, fever and cough and was found to have disease recurrence. He is now undergoing further chemotherapy which has again resulted in improvement in his symptoms.

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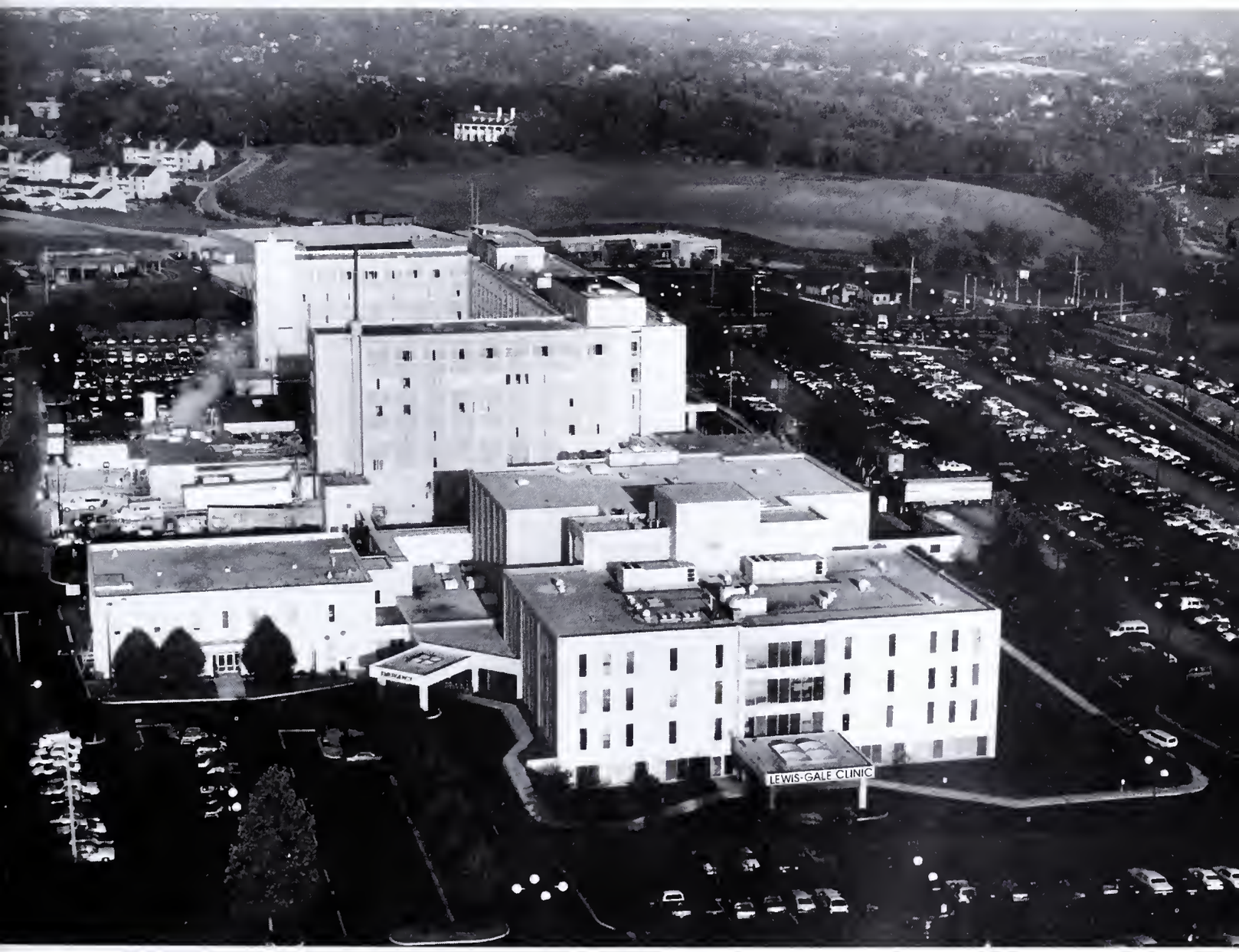

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ABSTRACTS

The following abstracts derive from a seminar on "Cardiac Arrhythmias Update 1990" sponsored by the Virginia Heart Institute on September 21, 1990, in Richmond.

Management of Ventricular Arrhythmias in the Post-CAST Era. Kenneth A. Ellenbogen, MD, Richmond.

Management of patients with nonsustained ventricular tachycardia and a decreased ejection fraction is of great importance to the general internist and cardiologist. These patients represent the majority of patients with ventricular arrhythmias. In these patients, the presence of nonsustained ventricular tachycardia and the concomitant decrease in left ventricular ejection fraction make them at significantly higher risk for life-threatening ventricular tachyarrhythmias in the future. Empiric antiarrhythmic drug therapy has not been shown to be effective in this patient population. The CAST trial, a recently performed multicenter placebo-controlled study of encainide, flecainide, and ethomozine for treatment of asymptomatic premature ventricular contractions in patients who survived a myocardial infarction, has shown a two- to three-fold increase in mortality in patients taking encainide or flecainide. This study was designed to test the hypothesis that suppression of PVCs measured by ambulatory Holter monitoring will lead to a decrease in cardiac mortality following myocardial infarction. This study emphasized the limitations and inadequacies of Holter monitoring and Class 1C antiarrhythmic drugs for treatment of ventricular ectopy. In addition, other studies such as meta-analyses of antiarrhythmic drug trials, have failed to show efficacy (prolonged survival) of 1A, 1B or phenytoin for Holter-guided treatment of premature ventricular contractions.

Based on these results, at the present time a strategy for treating patients with nonsustained VT and impaired left ventricular ejection fraction can be recommended. These patients should either undergo signal-averaged ECGs, and if positive, other evaluation, or undergo electrophysiologic testing with programed electrical stimulation to further define high-risk populations who would be more amenable to treatment with investigational antiarrhythmic drugs, amiodarone, or implantable devices.

Surgical Treatment of Arrhythmias. Ralph J. Damiano, Jr., MD, Richmond.

The modern era of cardiac arrhythmia surgery began in 1968 when Dr. Will Sealy performed the first surgical cure of a patient with Wolff-Parkinson-White syndrome. His initial pioneering efforts helped establish that a successful surgical approach must be based on an understanding of the mechanism of the arrhythmia, a knowledge of the anatomic substrate, and electrophysiologic guidance. While Wolff-Parkinson-White syndrome remains the gold standard against which other operations for arrhythmias are judge (morbidity and mortality with a surgical approach to Wolff-Parkinson-White syndrome approaching 0%), many new arrhythmias have recently become amenable to surgical treatment. In the last ten years, successful operations have been proposed for ectopic atrial tachycardia, atrioventricular node reentry, atrial flutter and fibrillation, inappropriate sinus node tachycardia, and ventricular tachycardia and fibrillation.

One of the most significant developments has been the introduction of computerized intraoperative mapping systems. By recording data from multiple areas of the heart simultaneously, it allows the electrophysiologist and surgeon to identify rapidly the location of the arrhythmogenic focus. At the Medical college of Virginia, we utilize a sophisticated mapping system that allows us to obtain 127 electrograms simultaneously. With interactive software, these data are quickly reviewed and then displayed as color-coded activation maps of the heart. These maps graphically depict the origin of the arrhythmia. This mapping system not only allows a raid identification of the abnormal area, but has allowed us to better understand the mechanisms of many of the atrial and ventricular arrhythmias that we are treating. It has allowed us to treat some unusual nonischemic ventricular tachycardias that we would be hesitant to bring to the operating room without such a system.

One of the most exciting areas in the treatment of supraventricular arrhythmias has been the recent introduction of procedures for the treatment of atrioventricular node reentry. This is an extremely common form of supraventricular tachycardia. While it usually responds to medical treatment, patients occasionally present with refractory tachycardia that in the past has required His bundle ablation and insertion of a pacemaker. This arrhythmia has been shown to originate from the atrioventricular node and perinodal tissue. A technique has recently been introduced by Dr. Cox and his colleagues at Washington University School of Medicine that allows for cryosurgical modification of the atrioventricular node. This procedure is carried out with a 3mm cryoprobe cooled to −60°C with internal expanding nitrous oxide. The atrioventricular node is encircled with small cryolesions that modify the arrhythmogenic substrate and cures the patients of their tachycardia. In the last year, we have treated six patients with cryosurgical modification and have had a 100% success rate.

The most exciting development in the treatment of ventricular tachycardia and ventricular fibrillation has been the introduction of the automatic implantable cardioverter defibrillator. This can be implanted in selected patients with ventricular tachycardia and fi-

brillation and has had a dramatic impact on patient survival. The device consists of a large generator that stores the defibrillation charge, sensing electrodes that tell the device when to deliver a shock, and patches that are placed around the heart through which the shock is administered. We implanted 59 devices at the Medical College of Virginia in 1989. The operative mortality with device implantation was 0%. Over 95% of patients who received these devices are alive as of last followup. This is a dramatic improvement over the 30-40% yearly mortality seen in these patients prior to the development of the defibrillator. We are currently involved at the Medical College of Virginia in clinical trials on the next generation of implantable defibrillators. These devices not only deliver defibrillation shocks, but also allow us to pace terminate tachycardias in certain instances. This requires a great deal less energy and is much more comfortable to the patient. These new devices represent a great technological advance and our initial experience has been very positive.

These abstracts derive from presentations at the 4th Annual Neurologically Impaired Individual Symposium: "The Legacy of Drugs," held in Richmond on March 16, 1990, under the sponsorship of Richmond Memorial and St. Mary's Hospitals and the Virginia Department of Health. The series is directed by Dr. Miriam W. Carmichael, Dr. Fritz E. Dreifuss, and Dr. James E. Etheridge, Jr.

Maternal Cocaine Use: Effect on Pregnancy, the Fetus, and the Newborn. Hallam Hurt, MD, Division of Neonatology, Albert Einstein Medical Center, Philadelphia, Pennsylvania.

Cocaine is easily absorbed across any membrane and crosses the placenta by rapid, simple diffusion. Adult clearance of cocaine and its metabolites requires several days; neonatal clearance, if exposed within 2–3 days of delivery, 4–6 days. Clinical reports of maternal cocaine use reflect an increased incidence of abortion, abruptio placentae, premature labor, preterm deliveries, meconium staining, intrauterine growth retardation and malformations. Genitourinary malformations and brain lesions were discussed. Conflicting clinical reports regarding occurrence of SIDS was presented. Cocaine and its metabolities are detectable in breast milk 12–60 hours after last use and can lead to tachycardia, tachypnea, hypertension, irritability, tremulousness, and convulsions in infants. Medical social risks to the newborn were noted to be loss of maternal instinct, poor parenting, abuse, and neglect. Passive inhalation of crack by infants has resulted in seizures and death.

Central Nervous System Effects of Prenatal Maternal Cocaine Use. Robert Clancy, MD, Associate Professor, Neurology and Pediatrics, University of Pennsylvania School of Medicine and the Children's Hospital, Philadelphia.

These mothers have little concern for their prenatal care, their own nutritional status during pregnancy and post-delivery, and have problems in bonding with their infants. Problems relating to maternal behavior that require consideration include: polydrug user, sexual activity and prostitution, and HIV status. Prenatal effects on the infants include prematurity, stillbirth, and abruptio placentae. The neonatal effects on the central nervous system may vary from severe to mild complications. The infants may demonstrate global asphyxia, intracranial hemorrhage, brain infarction, cerebral malformations, microcephaly and seizures. An acute intoxication syndrome consisting of irritability, tremulousness, hypertension, hyperreflexia, toxic seizures has been observed. Abnormal breathing patterns, neonatal behavioral disturbances, altered sleep architecture and maturation, and abnormal EEG's have been described. Post-natal effects include intoxication by passive exposure in the environment and breast milk exposure, sudden infant death syndrome, mental retardation, cerebral palsy and epilepsy. Subsequent development of learning disabilities and psychosocial problems was discussed.

Epilepsy, Pregnancy, and Birth Defect. Richard H. Finnell, PhD, Associate Professor and Director, Teratology Laboratory, Washington State University at Pullman.

The most recent epidemiologic and clinical data concerning the teratogenicity of anticonvulsant drugs have clearly established that the risk for producing a child with a congenital abnormality increases with the number of medications used to control the maternal seizure disorder. Furthermore, certain combinations of drugs appear to pose more serious risks than do others. Although the number of infants is limited, the data suggests that there are increased risks involved when combinations of oxidative metabolite-forming anticonvulsant drugs are used during pregnancy.

From the data reported in this study it appears to be possible to identify individuals at increased risk for phenytoin-induced congenital malformations by measuring the activity of the enzyme epoxide hydrolase in amniocytes. Using an animal model of phenytoin-induced teratogenesis, we manipulated the level of oxidative metabolites to which the fetuses were exposed by co-administering the P450 inhibiting anticonvulsant drug, stiripentol. This compound significantly reduced the incidence of phenytoin-induced congenital malformations, thereby confirming the hypothesis that oxidative metabolites are the critical factor mediating phenytoin teratogenesis.

continued over

Generic Drugs and Drug Interactions. Thomas R. Browne, MD, Associate Chief, Neurology, Veterans Administration Medical Center at Boston, Massachusetts.

Bioavailability of a generic drug is usually compared with bioavailability of a brand name drug by administering each drug to volunteers at different times under controlled conditions. FDA guidelines allow a generic product which produces widely varying bioavailability in comparison with brand name in some individuals to be approved as "equivalent" to brand name as long as the mean ±90% confidence interval values for AUC and other parameters falls within a range of ±20% in comparison with brand name and the "80%/80% rule" is met. These regulations do not insure that *all* individuals will receive the same amount of antiepileptic drug if they switch from brand name to generic antiepileptic drug or from one generic to another. *There is absolutely no scientific evidence to support the assertion that a ±20% range (or any other range) of variability in mean steady state serum concentration of antiepileptic drug can be tolerated safely by patients with epilepsy.* My experience indicates that an acceptable range of variability would be that *all* subjects must have AUC values with a generic drug which are within a +10% of the value with brand name drug. Current USP guidelines specify the average drug content of a tablet or capsule be 93% to 107% of the advertised drug content for most antiepileptic drugs. This range of variation in drug content is *in addition* to the range of variation in bioavailability allowable under FDA regulations and may amount to an additional 23% variation. At this time, generic substitution for Dilantin® and Tegretol® is not recommended.

Clinically significant drug interactions may occur when addition of a second drug has one or more of the following effects on the first drug: (a) decreased or increased absorption rate, (b) alteration of tissue binding, and/or (c) decreased or increased rate of biotransformation and/or excretion. Pharmacokinetic drug interactions may be bidirectional and one drug may have more than one type of pharmacokinetic drug interaction with the other drug.

The Legacy of Drugs. Peter Wolf, Judge of the Superior Court of the District of Columbia, Washington.

What has or will the drug abuse crisis leave us or our successors? What can we do to prevent this legacy from worsening? What can we do to remedy it?

Broader societal changes have to be considered; the drug problem cannot be viewed in isolation. To address the drug problem adequately we must deal with the dislocations these changes have produced; 1) the rights, equalities and liberties for women, as well as their economic needs; 2) the breakdown of the family, with resulting unwed and child mothers; 3) the tax revolt and decline in spending for social programs; 4) the proliferation of guns; 5) the lessening constraints of religion; 6) the awesome/power of television and its ability to make affluence appealing and seemingly attainable without effort; 7) the increasing ineffectiveness of public education; 8) the blame for problems on racism; 9) the scarcity of effective moral leadership in the nation; and 10) the divisiveness of the split along political party lines between the Presidency and Congress, leading to ineffectiveness in long-range planning.

Lawyers must find addition/better ways than the criminal law for fighting drug abuse. Legalization of drugs is not the answer. Physicians must be honest about drug treatments, and the search for medical treatment of addiction should be intensified. Those in ethics must face the issue of protecting the unborn and the newborn from drugs. In a world on fire with freedom nurtured by our flames, the United States cannot allow itself to become a smoldering ruin of addiction.

These are the three prize winning abstracts presented on Kinloch Nelson Medical Student Honors Day of the Medical College of Virginia/Virginia Commonwealth University on May 10, 1990. Faculty advisers are listed as coauthors.

Assignment of Neuroanatomical Correlates to the Vector Visual Evoked Potential Using Hemifield Stimulation. Lance M. Siegel (M-90), Juan Astruc, MD, Alfred L. Ochs, PhD, and Janet Thompson.

Visual evoked potentials are an accepted procedure for the evaluation of the integrity of the visual pathway. This procedure is generally performed in clinical practice by measuring the latency of responses recorded over the occipital pole. A principal positive wave occurring 100 msec following the pattern-reversal of a checkerboard stimulus (P100) is the only response routinely used clinically, since earlier and later evoked activity is poorly reproducible in conventional recordings. Using a three-channel vector recording montage (three electrode pairs placed across the skull at mutual right angles to each other on the scalp), visual evoked responses were reproducibly recorded both prior to and succeeding the P100 event. Since stimulation of a full retinal field tests two lateralized visual pathways, we used nasal and temporal hemi-field stimulation. Data were analyzed using three-dimensional computer graphics, and responses were found to be reproducible in time and space both within and among normal test subjects, producing peaks of activity at 55, 70, 100, 130, 160, 190 and 220 msec. A full-field response is shown to be the vector sum of the two individual hemi-field components, indicating the independence of these two lateral pathways. Spatial orientaion of consecutive electrical responses along the entire visual pathway provides a

description of the activity of the entire brain throughout the recording interval, allowing anatomical correlates to the assigned.

The Bone Marrow-Thymus Axis in Senescence. Kevin R. McCormick (M-93).

It has been postulated that the thymic hormones may act on lymphocyte precursors in the bone marrow and that the loss of thymic factors during senescence may be a contributing factor to the decreased cellular immune function. Tyan (1977) reported that aged mice exhibited an age-related decrease in T-cell progenitors. Haar et al (1988) showed that the ability of bone marrow cells of senescent mice to migrate to thymus supernatant in vitro is greatly reduced and is restored to youthful levels by the engraftment of thymic epithelial cells from neonatal mice. Hirokawa (1986) found that aged bone marrow had a greatly reduced ability to repopulate the thymus of an irradiated host. This study used an in vivo model to investigate the bone marrow-thymus axis in aged mice. Erythroid-depleted bone marrow cells from 3-month- and 24-month-old CBA (Thy 1.2) mice were given to irradiated AKR (Thy 1.1) mice and allowed to repopulate for 30 days. Flow cytometry analysis using mAb Thy 1.1 and Thy 1.2 revealed that the old bone marrow was deficient in its ability to repopulated the thymus. Subsequent experiments revealed that treatment of the old bone marrow with thymus supernatant, made from neonatal thymus cultures, could restore the thymus repopulating ability of these cells. These experiments indicate that prethymic stem cells in the bone marrow of aged mice are deficient in their ability to seed the thymus and that treatment of these cells with thymus products may restore their thymus repopulating ability.

Tissue Specific Expression of Chlordecone Reductase in Humans and the Induction of Related mRNAs in Rat and Rabbit Liver by Dexamethasone. Charles J. Winters (M-92), Peter A. Wyss, MD, and Philip S. Guzelian, MD.

The toxic organochlorine pesticide chlordecone (Kepone®) undergoes bioreduction to its alcohol metabolite in human liver. This conversion is controlled by a cytosolic, monomeric, and NADPH-dependent protein, termed chlordecone reductase (CDR), which may be of the aldo-keto reductase superfamily of xenobiotic metabolizing enzymes. The primary structure of human CDR has been elucidated by cDNA sequencing and was found to be similar to other aldo-keto reductases [Biochemistry (1990) 29, 1080-1087]. To more fully examine the expression of CDR in humans, we subjected total RNA from adult and fetal liver and kidney, along with RNA from adult lung and intestine to Northern analysis using the CDR cDNA. In the human tissue, we oberved detectable amounts of mRNA in adult liver and intestine only, providing further proof that expression of human CDR is species-specific and confined to the liver and intestinc. Although examples of aldo-keto reductase proteins have been found in a wide variety of tissues and species, little is known about the regulation or expression of these enzymes. Our recent experiments demonstrate the in vivo induction of a CDR-related mRNA in rat and rabbit liver following administration of the glucocorticoid dexamethasone. Further in vitro studies reveal that dexamethasone treatment of rat hepatocytes and Hep G2 cells could also induce hybridizable mRNA. Although the rat and rabbit liver proteins coded for by the CDR-related mRNA remain to be characterized, the induction of mRNA in Hep G2 raises the interesting possibility that human liver CDR is inducible by dexamethasone.

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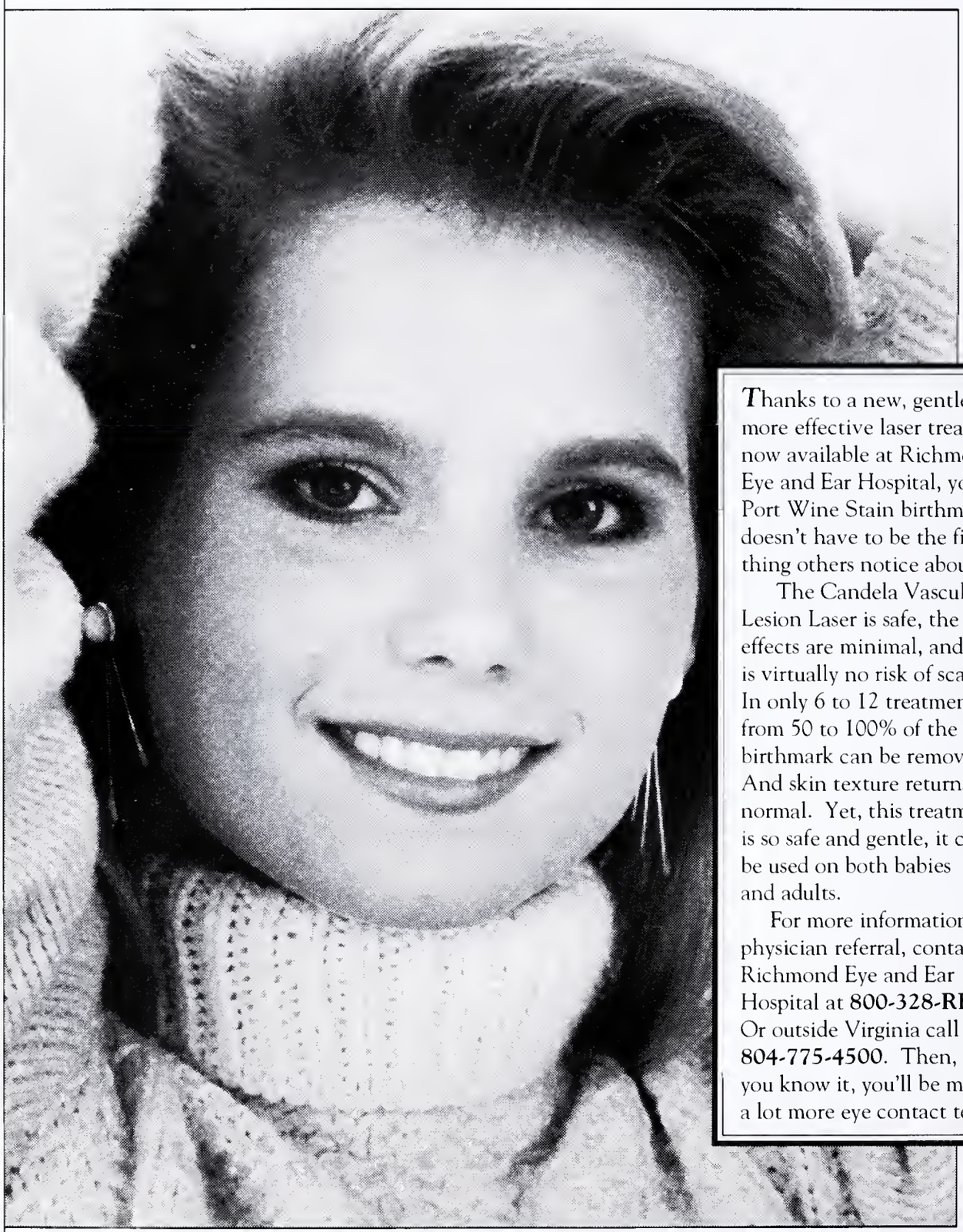

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In the Wake of Cruzan

In Virginia, Two Routes to Clear and Convincing Evidence

Nutrition and hydration. That was the clinical crux of the *Cruzan* case. No, said the United States Supreme Court to the parents of comatose Nancy Cruzan—no, you may not remove your daughter's nutrition/hydration support system because we do not have clear and convincing evidence that she would want it removed.

Clear and convincing evidence. That could be a "living will" of some kind. Two such surrogate decision-making instruments are now available to Virginians: the declaration form empowered by the Natural Death Act (1983) and the durable power of attorney for health care enabled by the Surrogate Decision-Making Statute (1989). Could they offer clear and convincing evidence in a nutrition/hydration withdrawal request? Yes, arguably, say Virginia attorneys. "Arguably" means that a law has not yet been tested in the courts but has a darned good chance of being upheld, as follows: 1) In *Cruzan*, the Supreme Court deferred, in effect, to the state legislatures to determine the mechanism and scope of these instruments. 2) Although neither the Act nor the Statute expressly authorizes the witholding/withdrawal of nutrition/hydration, neither precludes its use. In fact, the Circuit Court of Fairfax County, Virginia, in the case of *Hazelton v Powhatan Nursing Home*, ruled that the Natural Death Act permits a surrogate to request nutrition/hydration withdrawal.[1] And since the Surrogate Decision-Making Statute provides in its own words an alternative to other statutory and common law for making medical decisions, it can arguably be said to allow an appropriate surrogate to exercise such a constitutional right.[2]

In the wake of *Cruzan*, there's a clamor for living wills, and VA MED Q has assembled patient forms and physician guidelines for both Natural Death Act and durable power of attorney. All you have to do is fill out the coupon and put it in the mail. They are free of charge.

Both forms may be photocopied at will to give to your patients. Both can be completed without the assistance of an attorney, although they should be executed with all due care. Both are cognizant of Virginia law; they may not be viable in another state. Both require witnessing by two persons who are neither spouse nor blood relative of the signator. The durable power document asks for a notary seal; most banks provide notarization to their customers free of charge.

The original instrument should be kept in an accessible place—not a safety deposit box. The signator should also carry a wallet card indicating the existence of a surrogate decision-making instrument and should, if possible, inform all health care institutions upon entry of its existence. Copies should be given to 1) close relatives of the signator; 2) the signator's treating physician; and 3) any person designated to make surrogate decisions.

The Natural Death Act form is valid until revoked, and unless limited to a specific time period by the document itself, so too are the durable powers.[3] Either form can be revoked, but attorneys suggest that the revocation be in writing, in the manner of the original document, and that it be notarized. If the signator wants to designate a different surrogate or specify different treatment choices, the original document should be revoked and a new instrument written, signed, witnessed and notarized.

It is incumbent upon the treating physician to retain the document in the patient's medical record. Indeed, many physicians have begun routinely educating their patients to these documents and encouraging their use. They are no less a protection for the physician than for the patients. —A.G.

1. Personal communication, Julia Krebs-Markrich, August 15, 1990
2. Memorandum to medical clients and friends of Grad & Logan, PC, July 31, 1990
3. Personal communication from Michael P. Logan, November 19, 1990

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Dr. Barney (right) accepts the presidential seal from Dr. Owen.

The Passing Scene

DR. William H. Barney's term as President of the Medical Society of Virginia has ended, but admiration for his determination as well as his accomplishments will continue. In the mold of the recent President of the United States, he was a "great communicator." On two occasions he wrote to every physician in Virginia, members of the MSV as well as those who did not belong. And all of this in spite of health problems necessitating an angioplasty and later cardiac bypass surgery. During his term of office, the Society reinstated approximately 600 former members and added 300 new ones.

Now the stage is set for a new president, Dr. John A. Owen, Jr., of Charlottesville. John is an academician, a professor of medicine at the University of Virginia, and he will bring a fresh approach to the MSV presidency. The advent of an academician is timely; the most recent academician/president was Dr. McLemore Birdsong, who served in that post in 1965. Indeed, there have been only four full-time academicians as president since 1933; the others were Dr. James C. Flippen, 1933; Dr. Henry B. Mulholland, 1945; and Dr. Vincent W. Archer, 1954. All served on the faculty of the University of Virginia School of Medicine.

Dr. Owen also is a writer and an editor. He has for some years served as Associate Editor of VIRGINIA MEDICAL and has recently completed a term as Editor of the University of Virginia Medical Alumni News. He is a man of many talents. We welcome him.

E. L. K., JR.

VIRGINIA MEDICAL EDITORIAL

President Owen

THE NEW president of the Medical Society of Virginia is John Atkinson Owen, Jr., who was born the elder son of country doctor in Halifax County. One of his early memories is joining the family in addressing, by hand, all official communications of the South Piedmont Medical Society, of which the senior Dr. Owen was secretary-treasurer. It was always understood that he would follow in his father's footsteps.

And so he did, receiving his BS from Hampden-Sydney College in 1944 and his MD from the University of Virginia in 1948. At the University, he was elected to Alpha Omega Alpha and the Raven Society. He also won the prestigious Raven Award in 1948, an honor rarely conferred on a medical student.

After a rotating internship at Cincinnati General Hospital, Dr. Owen, who had joined the Navy's V-12 program in college, performed a year of active duty in Washington, DC. He next took two years of residency in internal medicine and endocrinology at the University of Virginia Hospital, then was recalled for another tour of military duty at the U. S. Naval Hospital, Portsmouth, and the U. S. Naval Dispensary on Guam. In 1954, he spent several interim months in family practice in Halifax with the late Dr. N. H. Wooding, then took two years of research training at Duke University under the late Dr. Frank L. Engel.

Dr. Owen's academic career began in 1956 with a faculty appointment to the Medical College of Georgia. After two years he moved to Washington, DC, as assistant chief of medicine at the VA Hospital there. In 1960 he joined the faculty of the University of Virginia and has held the rank of professor of internal medicine since 1970. He has served as chairman of the Drug and Pharmacy, Human Investigation, and Research Ethics Committees, recently edited the University of Virginia Medical Alumni News and has been head of the Division of Clinical Pharmacology and vice-chairman of the Department of Internal Medicine.

Outside the University his major interests have been in the fields of diabetes and drug therapy. A member of the American Diabetes Association, Endocrine Society and Southern Society for Clinical Investigation, he is currently conducting clinical trials of an aldose reductase inhibitor in the prevention and treatment of certain complications of diabetes. He was president of the U. S. Pharmacopeial Convention, Inc., 1975-80, and Editor in Chief of Hospital Formulary 1974-83. He has been a charter member of the Virginia Voluntary Formulary Board since its inception and has served under the last four governors.

As a long-time AMA member, Dr. Owen joined the Albemarle County Medical Society and the Medical Society of Virginia in 1960. He has served as president of the ACMS and as an MSV delegate since 1968. He became vice-councilor from the 7th District in 1976, councilor in 1983, and began his rise through the ranks to his current position in 1986. He has served on the Editorial Board, Scholarship, Ethics, and Aging Committees, and his service on the Physicians' Health and Effectiveness Committee may have spurred his selection as chairman of the University's Task Force on Drug and Alcohol Abuse and Education, which in 1986 began the process of counteracting the traditional peer pressures of the University student stereotype.

In 1952 Dr. Owen married the former Wanda Earle Reamy, born in Foneswood in Westmoreland County on the property where her Huguenot ancestors had lived since the 17th century. She obtained her R.N. from Johnston-Willis Hospital, Richmond, and is the mother of their two sons, John III and Ryland.

A long-time member and elder of Westminster Presbyterian Church, Dr. Owen relaxes by reading, gardening, haphazard tennis, and a primitive form of poker endemic among senior faculty in the University's School of Medicine.

Professional Courtesy

Is professional courtesy dead? While Dr. Turner's Letter to the Editor on page 6 is concerned with possible legal implications, the real issue seems to be whether or not professional courtesy continues or should continue to exist.

For as long ago as this writer can recall, and he was reared in a medical family and was himself graduated from medical school in 1936, physicians have provided professional services, without charge, to other physicians and their immediate families. Indeed, the physician was complimented that he/she had been chosen.

The situation has changed. Almost all physicians now possess hospital and medical insurance for themselves and their families. While it seems unlikely that anyone except those in the insurance company will ever completely understand insurance coverage, the physician treating another physician or his/her family usually receives remuneration, albeit not necessarily in the expected amount. The general rule now seems to be that the treating physician accepts payment as provided by the insurance company and does not seek further payment. This appears to be a gracious and logical approach. It may even be legal.

E. L. K., JR.

OBITUARY

• **Russell V. Bowers, MD,** long-time Richmond general practitioner; Medical College of Virginia, 1950; age 75; died October 5, 1990, in Deltaville.

• **Gloria G. Brennan, MD,** Fairfax pathologist; George Washington University School of Medicine, 1952; age 63; died March 3, 1990.

• **Alexander D. Crosett, MD,** Kilmarnock; Yale University School of Medicine, 1950; age 63; died October 7, 1990. A radiologist, Dr. Crosett was founder and president of the Chesapeake Mobile Imaging Service.

• **Jack W. Hall, MD,** Danville nephrologist; Medical College of Virginia, 1955; age 67; died October 23, 1990.

• **Thomas N. Hunnicutt, MD,** Newport News, retired specialist in pulmonary diseases; Medical College of Virginia, 1904; age 86; died July 3, 1990.

• **Orvin Clarence Jones, MD,** Newport News, retired ophthalmologist; Medical College of Virginia, 1927; age 89; died October 2, 1990.

• **Gordon E. Madge, MD,** Richmond; University of Maryland School of Medicine, 1953; age 63; died September 10, 1990. He was professor of pathology at the Medical College of Virginia.

• **John William Massey, MD,** Newport News internist; Medical College of Virginia, 1942; age 71; died October 8, 1990.

• **Lewis A. Micou, MD,** long-time general practitioner in Buena Vista; Medical College of Virginia, 1936; age 79; died October 25, 1990, in Virginia Beach where he had been living in retirement.

• **Fred F. Oast, MD,** Roanoke; University of Pennsylvania School of Medicine, 1923; age 85; died November 13, 1984. He was a pulmonary disease specialist.

• **Clyde Garvice O'Brien, MD,** general practitioner in Appomattox County; Medical College of Virginia, 1934; age 80; died on September 21, 1990. He retired a few years ago after serving the people of his native community for more than 40 years.

• **Wilmer Howard Paine, Jr., MD,** retired Charlottesville general practitioner; University of Virginia School of Medicine, 1930; age 86; died July 20, 1990.

• **Bruce Leonard Randolph, MD,** retired Richmond surgeon; Medical College of Virginia, 1926; age 91; died October 30, 1990.

• **John P. Snead III, MD,** Sperryville; University of Virginia School of Medicine, 1932; age 83; died October 7, 1990. Born in Rappahannock County, he practiced medicine there for 39 years.

• **Charles W. Steel, MD,** retired Suffolk internist; University of Virginia School of Medicine, 1936; age 80; died July 4, 1990.

• **Jack Langford Ulmer, MD,** retired Richmond neurosurgeon; Vanderbilt University School of Medicine, 1905; age 81; died November 22, 1990. He had been chief of neurological surgery at the McGuire Veterans Administration Medical Center.

• **George Lee Wilkinson, MD,** South Boston; Medical College of Virginia, 1953; age 63; died October 9, 1990. He retired recently after many years as a general practitioner.

Memoir of James N. Williams 1904-1990

By John Jay Krueger, MD

Dr. James N. Williams, retired psychiatrist, died September 30, 1990, in a nursing home in Virginia Beach.

"Dr. Jim," as he was affectionately known by Virginia Beach Rotarians, members of the Virginia Beach Good Shepherd Church, and many other friends, brought great credit to the medical profession through community involvement, generosity, and tireless service.

A native of Richmond, Dr. Jim was a graduate of Washington and Lee University (1926) and of the Medical College of Virginia (1930), where he served his residency in psychiatry from 1932 to 1935. In 1936 he went into private practice in Richmond, where he remained until 1941, when he entered the U.S. Navy, attaining the rank of captain and serving as a Navy psychiatrist for 30 years.

Always a leader, Dr. Jim was director of the Virginia Board of Mental Hygiene (1935-1941); chief of the neuropsychiatric service at the U.S. Naval Hospital in both Portsmouth and Newport News; director of the Portsmouth Area Counseling and Guidance Clinic (1950-1964) and the Atlantic Mental Hygiene Center in Virginia Beach (1960-1972). In 1961 he served on the advisory board of the Child and Family Service/ Travelers Aid in Portsmouth.

His memberships included the Virginia Beach Med-

ical Society, The Medical Society of Virginia, the American Medical Association, the Royal College of Psychiatrists in London, the American College of Physicians, and the Portsmouth Mental Hygiene Association. He had been president of the Virginia Beach Rotary Club and served on the executive board of the Virginia Beach Chamber of Commerce in 1966.

Survivors include his wife, Dorothy Behle Williams; a daughter, Dorothy M. Williams, Virginia Beach; a son, James N. Williams, Jr., Annandale; and a brother, Dr. Robert K. Williams of Richmond, also a psychiatrist.

Those who knew Dr. Jim during his long career will remember his good humor and dedicated service, which continued to the very end of his life on this earth.

Memoir of Harold Sisson 1912-1990

By Norman R. Tingle, Sr., MD

Harold E. Sisson died at his home in Warsaw, Virginia, on October 7, 1990, at the age of 78 after a long illness. He was a native of Richmond County, Virginia. He graduated from Warsaw High School in 1930, attended the College of William and Mary, and received his MD degree from George Washington University in 1938. He was an intern and resident physician at George Washington University Hospital for two years before entry into private practice.

Dr. Sisson was in the U.S. Army Medical Corps from 1940 to 1946, obtaining the rank of lieutenant colonel.

He practiced family medicine in Warsaw from 1946 until he retired due to poor health in 1976.

Dr. Sisson was a member of the George Washington Medical Society, the Medical Society of Virginia, the American Medical Association, and a past president of the Northern Neck Medical Association.

Upon Dr. Sisson's retirement, he received awards from the Richmond County Health Department, the Rotary International Service Club, and the Richmond County Ruritan Club. He was a life member of Totuskey Baptist Church in Haynesville, the Warsaw Mauman Lodge No. 332 AF&AM, the Westmoreland Royal Arch Chapter 41, and the Indian Creek Yacht and Country Club.

Survivors include two sisters, Mrs. Mary F. Urguhart of Alexandria and Mrs. Virginia H. Lokey of Spotsylvania.

Dr. Sisson was devoted to his profession and loyal to his patients, travelling many miles with daily house calls. His patients respected and loved him. Many lives were touched by this fine country doctor. His memory will be cherished by several generations.

Memoir of William Bickers 1908-1990

By Allen J. Award, MD, Henry A. Bullock, MD, and Philip L. Minor, MD

William M. Bickers—known by all as Billy—was raised in Richmond, attended William and Mary College, and graduated in medicine from the Medical College of Virginia. He then went to Boston for a three year residency in the very new specialty of obstetrics and gynecology. Upon completion of his residency Billy returned to Richmond and his alma mater to take the position of instructor in obstetrics and gynecology. A man who enjoyed teaching and sharing his knowledge, Dr. Bickers remained as instructor for eight years.

His charity work at Sheltering Arms and Retreat Hospitals and the Salvation Army Evangeline Booth Home and Hospital established his reputation for concern for the less fortunate and his love of his community. He was always trying to get new recruits for the specialty and help the newcomers establish themselves here.

A constant stream of original research work issued from his pen for publication. He read constantly and widely. He gave freely of his time to countless meetings of medical and hospital staff, and to churches and clubs.

In 1945 an opportunity arose which allowed him to continue teaching, this time in an area where good teachers of medicine were sorely needed. Dr. Bickers became visiting professor of obstetrics and gynecology at the American University of Beirut. He retained that post for a year.

Billy Bickers returned to private practice in downtown Richmond but still felt drawn to the East. In 1960 he returned to Lebanon as full professor and chairman of the Department of Obstetrics and Gynecology at the American University. He retained the Chair until retiring as Professor Emeritus in 1973. Upon retiring, Dr. Bickers resumed his private practice.

When he came home he lectured widely about the Middle East and published much original work on the problems encountered there.

Dr. Bickers was always a wise man. A wise man knows when the time has come to step down. Difficult as it was, William Bickers retired in 1986. A busy man throughout his life, Billy Bickers still found time for membership in many medical societies and the time to author three books. Two of the books are textbooks, *Gynecologist Therapy* and *Menorrhagia*. The third book, *Harem Surgeon*, tells of his experiences in the Middle East.

The skill and dedication of William Bickers did not go unnoticed. Dr. Bickers was the recipient of many awards, among them the Carmichael Award and the

Order of the Cedars. In 1981 he received the Distinguished Alumni Medallion and Honorary Award of the College of William and Mary.

In his retirement William Bickers combined his love of Virginia with his love of Lebanon by making generous contributions to the William and Mary School of Middle Eastern Studies. An endowment fund was also set up to broaden international knowledge and understanding of the Middle East.

Memoir of Thomas Frey 1934-1990

By M. Mendel Bocknek, MD

Those of us who knew Tom Frey, mourn his passing. Tom passed away on July 10, 1990, after a valiant fight against malignant melanoma.

A native of New York City, Dr. Frey earned his undergraduate degree from Cornell University and his medical degree from Northwestern University Medical School in 1958. Internship followed at Philadelphia General; a year of internal medicine residency at Bronx V.A. Hospital; and an ophthalmology residency at the University of Wisconsin Hospital in Madison. Prior to his ophthalmology residency, he served as a captain in the U.S.A.F. from 1960 to 1962.

In 1965, Tom, his wife Sue, and his first-born, Robert, moved to Northern Virginia, and Tom went into private practice at the Falls Church Medical Center. His rapport with children led him to take a fellowship with Dr. Marshall Parks in pediatric ophthalmology at Children's Hospital in Washington DC. From 1969 until his untimely death, he practiced pediatric ophthalmology in Northern Virginia.

Tom's work as a leader in ophthalmology increased as he matured. For 25 years he was a member of the board of directors of the National Children's Eye Care Foundation and was president in 1986.

THOMAS FREY, MD
1934-1990

His American Academy of Ophthalmology activities included associate secretary for federal legislation, Secretariat for Governmental Affairs, 1980-1987; regional director, Board of Councillors 1987; chairman, Board of Councillors 1988; director-at-large on the Academy Board 1988-1990. While serving in these capacities, Tom continually urged his colleagues in the grass-roots to let the Academy know how they felt on issues affecting patient care and ophthalmology.

From 1976-1980, Dr. Frey was on the Executive Committee of the Northern Virginia Academy of Ophthalmology, and served as its president from 1980-1982. Following this he was awarded the prestigious Haggerty Award by his peers in the Academy "for meritorious service to Virginia ophthalmology." From May 1988-May 1990 he was president of the Virginia Society of Ophthalmology. General medical affiliations were with the Fairfax County Medical Society and The Medical Society of Virginia.

Dr. Frey was on the staffs of the Fairfax, Fair Oaks, Arlington and Alexandria Hospitals, and directed the Pediatric Ophthalmology Clinic at Fairfax Hospital. He served as associate clinical professor at the George Washington University School of Medicine; as consultant to the Northern Virginia Training School for the Mentally Retarded; and particularly enjoyed his role as staff Ophthalmologist to the Washington Redskins football team.

Tom was a strong family man. He was an active member of the Olam Tikvah Synagogue in Fairfax and thoroughly enjoyed the many family-oriented religious holidays.

Immediate family survivors include his wife Sue of Annandale, whom he married in 1958; three children, Robert of New Carrolton, Maryland, Erin of Annandale, Virginia, and Heather Frey Zabrouski of Woodbridge, Virginia; his father, Theodore Frey of Fairfax; and his sister, Margery Small of Valley Stream, New York.

Tom's family requests that should his friends wish to perpetuate his memory, contributions in Tom's name may be made to the National Children's Eye Care Foundation, Cleveland, Ohio, and sent to the National Children's Eye Care Foundation, c/o George R. Beachamp, MD, 1 Clinic Center, 9500 Euclid Avenue, Cleveland OH 44195-5001.

Tom's kindness, bright outlook and spontaneous New York humor were well-known trademarks of this lovely man. With this God-given attribute, he made people of all stations in life feel comfortable about themselves. He often said "We did good." Tommy, "*You* did good," and your colleagues are enriched with having had the privilege of knowing and working with you. You will be long remembered.

Compiled from information from the AAO, VSO, Fairfax County Medical Society, and fond memories of 25 years with Tom.

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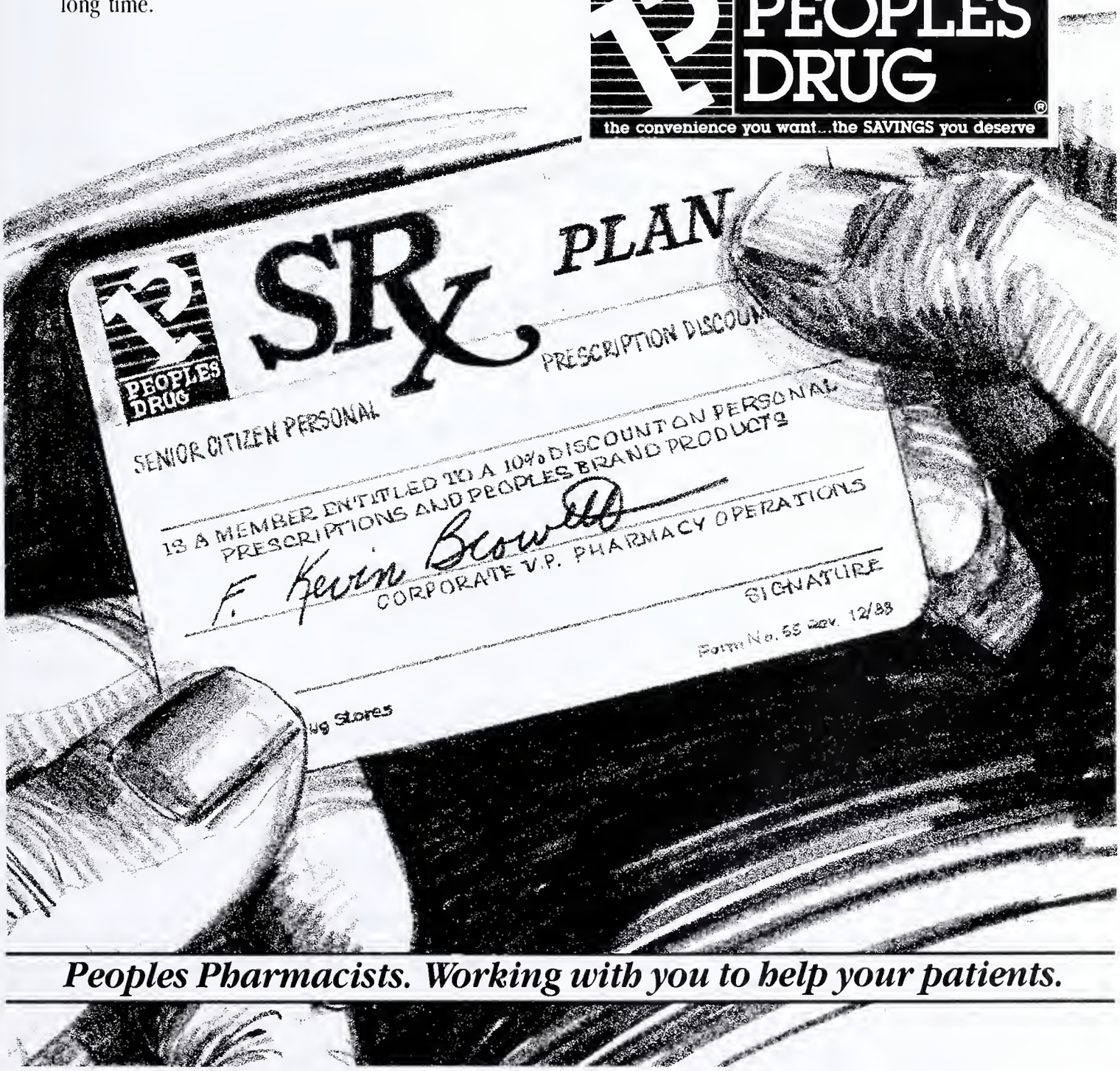

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MEETINGS

February 1-3
Annual Meeting of the Virginia Neurological Society, the Homestead, *Hot Springs*. Donna Scott, 804-353-2721.

February 10-14
1991 Scientific Assembly of the Virginia Chapter, American College of Emergency Physicians, the Homestead, *Hot Springs*. 18 credit hrs. Gwen Messler Harry, 804-966-5966.

February 23-March 2
5th Annual Current Innovations in the Practice of Gastroenterology, (Georgetown University), *Snowmass Village, Colorado*. CME Office, 202-687-8735.

March 1-3
Virginia Pediatric Conference '91: Annual Meeting of the Virginia Chapter, American Academy of Pediatrics and the Virginia Pediatric Society, Boar's Head Inn, *Charlottesville*. 804-643-8130.

March 4-7
Alton D. Brashear Postgraduate Course in Head/Neck Anatomy (Medical College of Virginia), *Richmond*. Dr. Hugo R. Seibel, 804-786-9624.

March 7-8
14th Annual Symposium on Mental Health and the Law (University of Virginia/Institute of Law, Psychiatry & Public Policy/Virginia Department MHMRSA/Office of Attorney General), Marriott Hotel, *Richmond*. Carolyn Engelhard, 804-924-5435.

March 14-16
Brain Chemistry and Behavior: Advances in PET and SPECT Imaging (Johns Hopkins), *Baltimore*. 18 credit hrs. Fee: $440. CME Office, 301-955-3839 or -8582.

March 14-17
11th Edition: Clinical Electrocardiography (Eastern Virginia Medical School), *Washington DC*. 24 credit hrs. Fee: $475. CME Office, 804-446-6140.

March 21-22
Clinical Care of the Patient with HIV Infection (Johns Hopkins), *Baltimore*. 14 credit hrs. Fee: $300. CME Office, 301-955-2959.

March 23-24
Pediatric Advanced Life Support Provider Course (Mary Washington Hospital), *Fredericksburg*. Limited to 24 students. Dr. Melville G. Wright III, 703-899-1326.

April 3
Louise Obici Memorial Hospital 38th Annual Clinical Conference, National Guard Armory, *Suffolk*. George J. Carroll, MD, 804-934-4000.

Pediatric Advanced Life Support Provider Course
March 23-24, 1991
October 12-13, 1991
Mary Washington Hospital
Fredericksburg, VA

American Heart Assoc. Approved
AMA Cat. 1 Credits
Limited to 24 students
Contact:
Melville G. Wright III, M.D.
Course Director
Lana King, RN
Course Coordinator
Mary Washington Hospital
Fredericksburg, VA 22401
(703)899-1326

April 4-7
2nd Annual Combined Conference on Breast Cancer (Eastern Virginia Medical School), *Williamsburg*. CME Office, 804-446-6140.

April 5-7
11th Annual Clinical Concerns in Primary Care (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

April 8-13
18th Annual Pediatric Trends (Johns Hopkins), *Baltimore*. 45 credit hrs. Fee: $575. CME Office, 301-955-2959.

April 10-13
Comprehensive Review of Clinical Obstetrics/Gynecology (Georgetown University), *Washington DC*. CME Office, 202-687-8735.

April 11-12
Pediatric Nutrition (Eastern Virginia Medical School), *Virginia Beach*. CME Office, 804-446-6140.

April 12-13
Otolaryngology/Ophthalmology Update for the Primary Care Physician (Eastern Virginia Medical School), *Williamsburg*. CME Office, 804-446-6140.

April 12-14
11th Annual Ophthalmology Conference: the Diabetic Eye (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

Continued page 60

April 18-21
6th Annual Review Course in Reproductive Endocrinology/ Assisted Reproduction (Eastern Virginia Medical School), *Williamsburg*. CME Office, 804-447-6140.

April 18-21
14th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *Washington DC*. 22 credit hrs. Fee: $425. CME Office, 804-446-6140.

April 19-21
13th Annual Conference on Emergency Medicine for the Primary Care Physician (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

April 25-27
Advances in Hip and Knee Arthroplasty (Johns Hopkins), *Williamsburg*. 18 credit hrs. Fee: $575. CME Office, 301-955-2959.

April 26-28
26th Annual Pediatric Springfest (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

May 3-5
Annual Meeting of the Virginia Surgical Society, *Williamsburg*. Irving L. Kron, MD, 804-924-2158.

May 9-12
Annual Meeting of the Virginia Chapter, American College of Radiology, *Williamsburg*. Patricia R. Berry, 703-669-8312.

May 10-11
Annual Meeting of the Virginia Society of Ophthalmology, Hyatt House, *Reston*. Donna Scott, 804-353-2721.

May 16-17
Pediatric Allergy/Immunology for the Practitioner (Johns Hopkins), *Baltimore*. Fee: $195. CME Office, 301-955-2959.

May 17-18
Annual Meeting of the Virginia Society of Otolaryngology/ Head and Neck Surgery, Omni Hotel, *Norfolk*. Donna Scott, 804-353-2721.

May 25-27
12th Annual Conference on Urogynecology and Pelvic Surgery (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

June 3-7
7th EVMS Family Medicine Review Course (Eastern Virginia Medical School), *Virginia Beach*. 42 credit hrs. Fee: $450. CME Office, 804-446-6140.

June 6-9
15th Annual Postgraduate Course on Rehabilitation of the Brain-Injured Adult and Child (Medical College of Virginia/ VCU), *Williamsburg*. CME Office, 804-786-0494.

June 14-16
Annual Meeting of the Virginia Allergy Society, Ramada Oceanfront Inn, *Virginia Beach*. Donna Scott, 804-353-2721.

June 20-23
15th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *San Francisco, California*. 22 credit hrs. Fee: $450. CME Office, 804-446-6140.

June 28-30
9th Summer Symposium in Internal Medicine (Eastern Virginia Medical School), *Williamsburg*. 12 credit hrs. CME Office, 804-446-6140.

July 19-21
13th Annual Pediatric Primary Care Conference: Pediatrics at the Beach (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

July 26-28
Practical Internal Medicine Conference (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

August 8-11
15th Annual Summer Retreat: Practical Issues in Primary Care (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

August 16-18
7th Annual Conference on Primary Care of the Female Patient (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

September 21-23
Annual Meeting of the Virginia Vascular Society, Tides Lodge, *Irvington*. Dr. Walter H. Graham, 804-596-7631.

October 12-13
Pediatric Advanced Life Support Provider Course (Mary Washington Hospital), *Fredericksburg*. Limited to 24 students. Dr. Melville G. Wright III, 703-899-1326.

NEW MSV MEMBERS

Alexandria Medical Society

Michael C. Trahos, DO, Family Practice, 4600 King Street, Alexandria VA 22302

Arlington County Medical Society

Neil Levin, MD, Cardiology, 611 South Carlin Springs Road, Arlington VA 22204

Augusta Highland Medical Society

Omar J. Beiler, MD, Family Practice, 517 Cambridge Drive, Stuarts Draft VA 24477

Dennis L. Hatter, MD, Family Practice, 113 First Street, Stuarts Draft VA 24477

Chesapeake Medical Society

Nancy M. Welch, MD, Public Health, 748 Battlefield Boulevard, Chesapeake VA 23320

Fairfax County Medical Society

Amer Z. Al-Juburi, MD, Urology, 10721 Main Street, #3400, Fairfax VA 22030

Patrick J. Byrne, MD, Oncology, 4660 Kenmore Avenue, Alexandria VA 22304

Preston C. Calvert, MD, Neurology, 7799 Leesburg Pike, Falls Church VA 22043

Tet Wei Chan, MD, Internal Medicine, 2839 Kalmia Lee Court, Falls Church VA 22042

David M. Dunning, MD, Hematology, 4660 Kenmore Avenue, Alexandria VA 22304

Anne Rose Eapen, MD, Internal Medicine, 3299 Woodburn Road, Annandale VA 22003

Denise M. Fraser, MD, General Surgery, 3301 Woodburg Road, Annandale VA 22003

Steven W. Musto, MD, Critical Care Medicine, 2230 George C. Marshall Drive, Falls Church VA 22043

Phillip L. Pearl, MD, Child Neurology, 7799 Leesburg Pike, #444 South, Falls Church VA 22043

Cary C. Schwartzbach, MD, Orthopedic Surgery, 8346 Traford Lane, #B-4, Springfield VA 22152

Marla L. Shuman, MD, Pulmonary Medicine, 8318 Arlington Boulevard, Fairfax VA 22031

John E. Threlfall, MD, Anesthesiology, 9124 Vendome Drive, Bethesda MD 20817

Fredericksburg Area Medical Society

Larry T. Hegland, MD, Anesthesiology, 2101 Fall Hill Avenue, Fredericksburg VA 22401

Andrew M. Margileth, MD, Pediatrics, 1438 Kenmore Avenue, Fredericksburg VA 22401

Charles G. Penick, Jr., MD, Emergency Medicine, 119 Caroline Street, Fredericksburg VA 22401

Loudoun County Medical Society

George S. Poehlman, MD, Family Practice, 209 Old Waterford Road, Leesburg VA 22075

Norfolk Academy of Medicine

John S. Alspaugh, MD, Plastic Surgery, 400 West Brambleton Avenue, Norfolk VA 23510

Robert D. Lehman, MD, Pediatrics, 110 Kingsley Lane, Norfolk VA 23505

Gary M. Mason, MD, Obstetrics/Gynecology, 112 West Plume Street, Norfolk VA 23510

Joan H. Rose, MD, Hand Surgery, 6160 Kempsville Circle, Norfolk VA 23502

Patrick Henry Medical Society

Matthew J. Mlot, MD, Gastroenterology, 312 Fairy Street, Martinsville VA 24112

Portsmouth Academy of Medicine

Sandra S. Baucom, MD, Pediatrics, 3620 Clifford Street, Portsmouth VA 23707

John K. Y. Chacko, MD, General Surgery, 3640 High Street, Portsmouth VA 23707

Nathan P. Goldin, MD, Urology, 720 Rodman Avenue, Portsmouth VA 23707

Robert B. Hansen, MD, Neurology, 300 North Street, Portsmouth VA 23704

Ernest A. Murden, Jr., MD, Otolaryngology, 430 Jamestown Avenue, Portsmouth VA 23704

Javaid A. Perwaiz, MD, Obstetrics/Gynecology, 3003 Churchland Boulevard, Chesapeake VA 23321

Jeanne L. Shepherd, MD, Otolaryngology, 508 Patric Street, Portsmouth VA 23707

Prince William County Medical Society

Michael D. Perez, MD, Family Practice, 9202 Centerville Road, Manassas VA 22110

Richmond Academy of Medicine

Charles G. Alty, MD, General Surgery, 2315 Essex Road, Richmond VA 23228

Alan J. Appley, MD, Neurological Surgery, Box 631, MCV Station, Richmond VA 23298

Nadua P. Blanchet, MD, Plastic Surgery, 9220 Forest Hill Avenue, Richmond VA 23235

Sallie S. Cook, MD, Pathology, 2412 Hampden Row, Rockville VA 23146

Ralph J. Damiano, Jr., MD, Cardiovascular Diseases, Box 68, MCV Station, Richmond VA 23298

Richard H. Hoffman, MD, Family Practice, 2500 Pocoshock Place, Richmond VA 23235

Gary K. Lofland, MD, Cardiovascular Surgery, Box 68, MCV Station, Richmond VA 23298

James W. Stone, Jr., MD, Anesthesiology, 2230 Galloway Terrace, Midlothian VA 23113

Ronald E. Whitley, MD, General Surgery, 1401 Johnston-Willis Drive, Richmond VA 23235

continued page 65

"Our Concern Be Peace Of Mind."

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Arthur E. Kelley, M.D.


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Don L. Weston, M.D.


Orren LeRoyce Royal, M.D.


G. Paul Hlusko, M.D.


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Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:
• Discontinue Ceclor in the event of allergic reactions to it.
• Prolonged use may result in overgrowth of nonsusceptible organisms.
• Positive direct Coombs' tests have been reported during treatment with cephalosporins.
• Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
• Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
• Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
• Hypersensitivity reactions have been reported in about 1.5% of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of **serum-sickness-like** reactions have been reported with the use of Ceclor. These are characterized by findings of erythema multiforme, rashes, and other skin manifestations accompanied by arthritis/arthralgia, with or without fever, and differ from classic serum sickness in that there is infrequently associated lymphadenopathy and proteinuria, no circulating immune complexes, and no evidence to date of sequelae of the reaction. While further investigation is ongoing, **serum-sickness-like** reactions appear to be due to hypersensitivity and more often occur during or following a second (or subsequent) course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults with an overall occurrence ranging from 1 in 200 (0.5%) in one focused trial to 2 in 8,346 (0.024%) in overall clinical trials (with an incidence in children in clinical trials of 0.055%) to 1 in 38,000 (0.003%) in spontaneous event reports. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy; occasionally these reactions have resulted in hospitalization, usually of short duration (median hospitalization = two to three days, based on postmarketing surveillance studies). In those requiring hospitalization, the symptoms have ranged from mild to severe at the time of admission with more of the severe reactions occurring in children. Antihistamines and glucocorticoids appear to enhance resolution of the signs and symptoms. No serious sequelae have been reported.
• Stevens-Johnson syndrome, toxic epidermal necrolysis, and anaphylaxis have been reported rarely. Anaphylaxis may be more common in patients with a history of penicillin allergy.
• Gastrointestinal (mostly diarrhea): 2.5%
• Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
• As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
• Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
• Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1% and, rarely, thrombocytopenia and reversible interstitial nephritis.

Abnormalities in laboratory results of uncertain etiology.
• Slight elevations in hepatic enzymes.
• Transient lymphocytosis, leukopenia, and, rarely, hemolytic anemia and reversible neutropenia.
• Rare reports of increased prothrombin time with or without clinical bleeding in patients receiving Ceclor and Coumadin concomitantly.
• Abnormal urinalysis; elevations in BUN or serum creatinine.
• Positive direct Coombs' test.
• False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

PA 8791 AMP [021490LRI]

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VIRGINIA MEDICAL QUARTERLY

APRIL/MAY/JUNE 1991 VOLUME 118 NUMBER 2 ISSN 1052-4231

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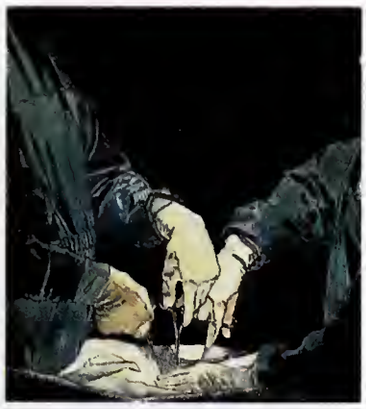

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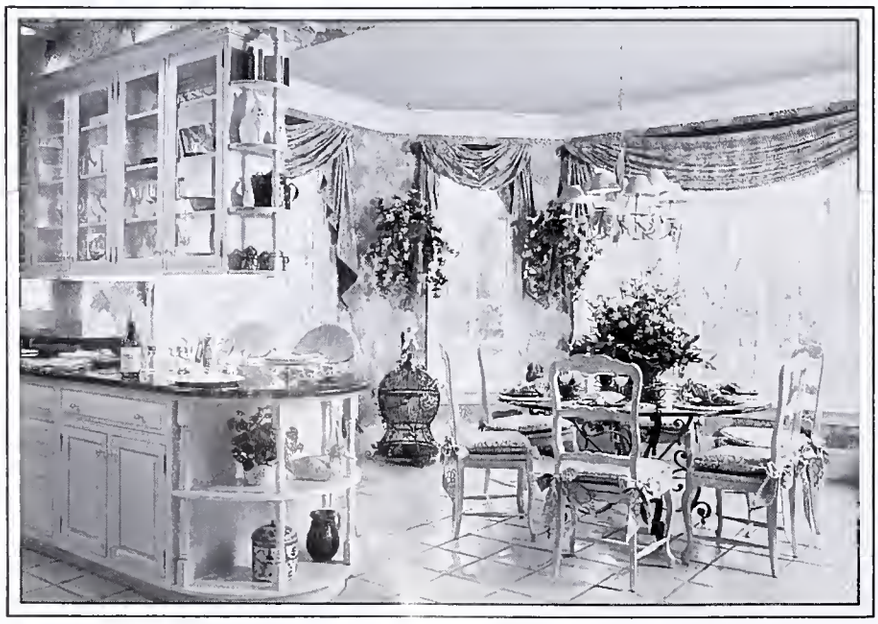

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VIRGINIA MEDICAL QUARTERLY (ISSN 1052-4231) is owned, published, and © copyrighted 1991 by The Medical Society of Virginia. It is issued in January (Winter), April (Spring), July (Summer), and October (Autumn). Second-class postage paid at Richmond, Virginia. Yearly subscription rate: $24 domestic, $30 foreign; single copies, $6. POSTMASTER: Send address changes to VMQ, 4205 Dover Road, Richmond VA 23221.

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On the cover: Sitting in the wheelchair is 103-year-old Joseph Duckworth, WWI veteran who is a patient in the 120-bed Nursing Home Care Unit at the McGuire Veterans Affairs Medical Center in Richmond. Shown with him is a geriatric nurse practitioner . . . The original art of surgeons' hands appears courtesy of the *Bulletin* of the American College of Surgeons, where the artist, Fred Semmler, is on staff.

THERAPY THAT MAY BE AS SILENT AS HYPERTENSION ITSELF

VASOTEC is generally well tolerated and not characterized by certain undesirable effects associated with selected agents in other antihypertensive classes.

VASOTEC is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

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For a Brief Summary of Prescribing Information, please see the last page of this advertisement.

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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor

Warnings: ***Angioedema:*** Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and appropriate therapy and monitoring should be provided until complete and sustained resolution of signs and symptoms has occurred. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL) and/or measures necessary to ensure a patent airway, should be promptly provided.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed, caution should be observed when initiating therapy (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis. Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Fetal/Neonatal Morbidity and Mortality: ACE inhibitors, including VASOTEC, can cause fetal and neonatal morbidity and mortality when administered to pregnant women.

Enalapril crosses the human placenta. When ACE inhibitors have been used during the second and third trimesters of pregnancy, there have been reports of hypotension, renal failure, skull hypoplasia, and/or death in the newborn. Oligohydramnios has also been reported, presumably representing decreased renal function in the fetus; limb contractures, craniofacial deformities, hypoplastic lung development and intrauterine growth retardation have been reported in association with oligohydramnios. Patients who do require ACE inhibitors during the second and third trimesters of pregnancy should be apprised of the potential hazards to the fetus, and frequent ultrasound examinations should be performed to look for oligohydramnios. If oligohydramnios is observed, VASOTEC should be discontinued unless it is considered life-saving for the mother.

Other potential risks to the fetus/neonate exposed to ACE inhibitors include: intrauterine growth retardation, prematurity, patent ductus arteriosus, fetal death has also been reported. It is not clear, however, whether these reported events are related to ACE inhibition or the underlying maternal disease. It is not known whether exposure limited to the first trimester can adversely affect fetal outcome.

Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion.

Enatapril has been removed from the neonatal circulation by peritoneal dialysis and theoretically may be removed by exchange transfusion, although there is no experience with the latter procedure.

There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril, but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day but not at 30 mg/kg/day (50 times the maximum human dose).

If VASOTEC is used during pregnancy or if the patient becomes pregnant while taking VASOTEC, the patient should be apprised of the potential hazards to the fetus.

Precautions: *General: Impaired Renal Function.* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions.*)

Cough: Cough has been reported with the use of ACE inhibitors. Characteristically, the cough is nonproductive, persistent and resolves after discontinuation of therapy. ACE inhibitor-induced cough should be considered as part of the differential diagnosis of cough.

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients: Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions. Hypotension. Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release. The antihypertensive effect of VASOTEC® (Enalapril Maleate, MSD) is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy: Pregnancy Category D. See WARNINGS, *Fetal/Neonatal Morbidity and Mortality.*

Nursing Mothers: Enalapril and enalaprilat are detected in human milk in trace amounts. Caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use. Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION. The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE. The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*), pulmonary embolism and infarction; pulmonary edema; rhythm disturbances including atrial tachycardia and bradycardia, atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis (hepatocellular [proven on rechallenge] or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth.

Musculoskeletal: Muscle cramps.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritus, alopecia, flushing, diaphoresis.

Special Senses: Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), impotence.

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgia/arthritis, myalgia, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Fetal/Neonatal Morbidity and Mortality: In infants exposed *in utero* to ACE inhibitors the following adverse experiences have been reported: Fetal and neonatal death, renal failure, hypoplastic lung development, hypotension, hyperkalemia, skull hypoplasia, limb contractures, craniofacial deformities, intrauterine growth retardation, prematurity and patent ductus arteriosus. (See WARNINGS, *Fetal/Neonatal Morbidity and Mortality.*)

Clinical Laboratory Test Findings: Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASDTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit. Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g% and 1.0 vol%, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolysis have been reported in patients with G6PD deficiency.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486. J9VS61R2(824)

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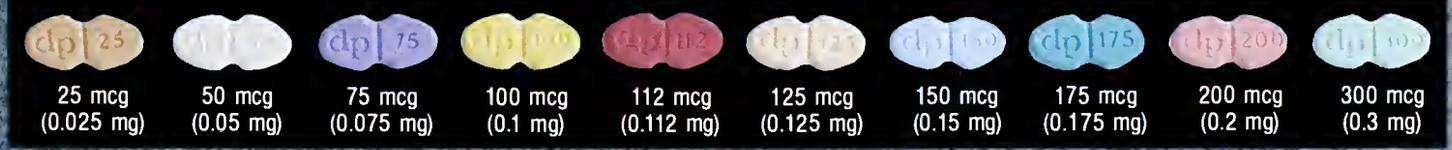

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$Medicare$: Reductions, reforms, experiments

The Administration continues to target Part B (physician payments) for cuts in the FY '92 budget. The impetus is continued double-digit increase in Part B expenditures. During the 1985-1989 period, Part B payments rose at an average annual rate of 15.5%. In the same period, Part A expenditures (payments to hospitals and nursing homes) increased only 4.6% per annum, due in large part to implementation of DRG payments.

In 1992, expect further reductions in reimbursement for 1,400 so-called "overvalued" procedures, mostly surgical. Additional cuts are expected in payments for pathological, radiological and anesthesiology services. The only bright spot will be increased reimbursement for primary care (cognitive) services.

Payment Reform

Medicare officials are looking seriously at revising definitions of office visit codes. The major change would make time a major factor in determining appropriate code levels. The scale might range from 5 minutes for a brief office visit to 60 minutes for a comprehensive physical examination. While such a system might simplify coding and billing, it could be inaccurate for the slower, more methodical practitioner.

Revised projections of the impact from RBRVS indicate that surgery payments may not decrease quite as much as initially estimated. Conversely, general and family practitioners may do somewhat better than originally projected. Physicians in rural areas are expected to see an improvement in their share of the Medicare pie, particularly as compared with expensive urban areas (such as NYC and Los Angeles), which will receive substantial reductions in payment levels.

Experimental Procedures

HCFA will publish this spring in the *Federal Register* a proposed new coverage policy rule. This rule determines how Medicare decides whether and how to pay for new medical and surgical procedures. Currently, individual Medicare carriers make many such decisions themselves. Carriers often consider new procedures to be "experimental" or "investigational" long after they have gained widespread acceptance among physicians.

Publication of the proposed rule will provide physicians with a golden

opportunity to submit written comments to HCFA regarding the form such a revised rule should take. The recent success in effecting change in proposed physician laboratory rules was the direct result of over 50,000 written comments received by HCFA from physicians.

—DAVID J. LATHAM

Attorney Latham practices out of a Richmond office as an associate of the Alexandria law firm of Grad & Logan.

Powerful new regs target service to disabled patients

Legal counsel alerts Medical Society of Virginia members to a new set of federal regulations of enormous significance to those who deliver health care. They derive from the Americans with Disabilities Act of 1990, which attorneys say is equal in scope and power to the Civil Rights Act of 1964.

Physicians' offices are specifically targeted by Title III of the Act as "public accommodations" (and so, more expectedly, are medical clinics and hospitals) and may not discriminate in the provision of goods, services, or facilities against any person on the basis of disability. This prohibition applies to any entity that owns, leases from, leases to, or simply operates a place of public accommodation.

Architectural access is key. Does your practice site have wide doorways, ramps, grab bars? The Act as written requires retrofitting if it is readily achievable, and if you undertake any renovation or build a new facility, these regulations will be looking over your shoulder at the plans.

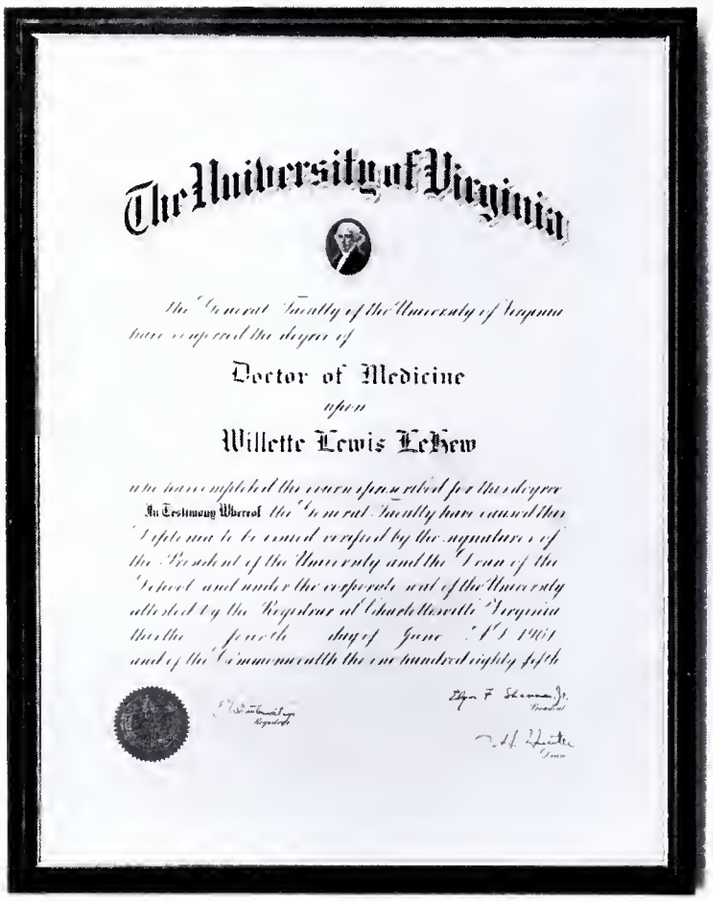
Also mandated are "auxiliary aids and services" to the disabled, such as readers for the blind and interpreters for the deaf. You will do well to delegate certain staff persons to act as needed in these capacities.

Most importantly, the Act emphatically states that you may not deny your professional services to a disabled person, and its three-part definition of a disabled person is comprehensive, as follows: 1) He or she has a physical or mental impairment substantially limiting his or her ability to walk, see, hear, speak, learn, or participate in any of life's other major activities. 2) He or she has a history of impairment. 3) He or she is regarded by others as being impaired.

The Americans with Disabilities Act will become enforceable on January 26, 1992. Enforcement will be through both private right of action and the U.S. Attorney General.

The information for this notice was supplied by Mark S. Hedberg of the law firm of Hunton & Williams, Richmond. For details pertinent to your situation, consult your lawyer.

INDEX TO ADVERTISERS



The University of Virginia

The General Faculty of the University of Virginia have conferred the degree of

Doctor of Medicine

upon

Willette Lewis LeHew

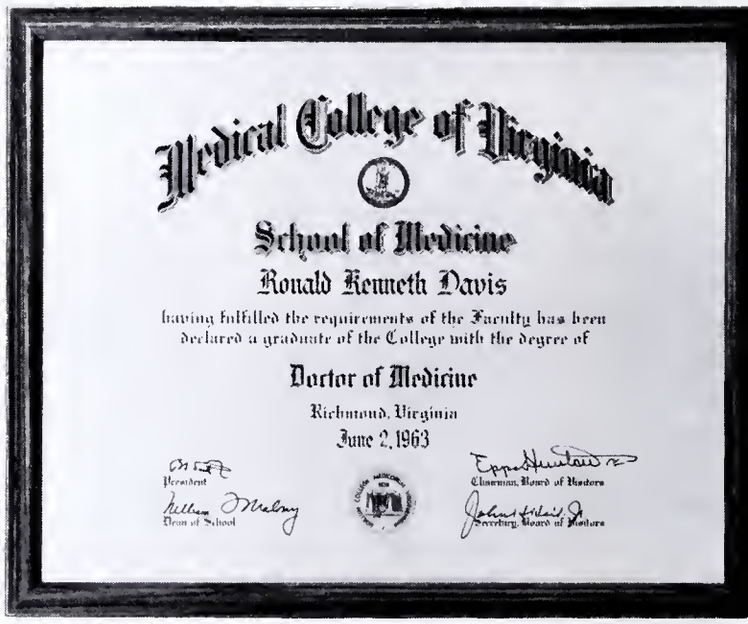
who has completed the course prescribed for this degree.

In Testimony Whereof the General Faculty have caused this Diploma to be issued verified by the signatures of the President of the University and the Dean of the School and under the corporate seal of the University attested by the Registrar at Charlottesville Virginia this the fourth day of June A.D. 1964 and of the Commonwealth the one hundred eighty fifth

Elgin F. Shannon Jr. President

M. Hunter Dean

Director: Willette L. LeHew, M.D., Norfolk



Medical College of Virginia

School of Medicine

Ronald Kenneth Davis

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Doctor of Medicine

Richmond, Virginia

June 2, 1963

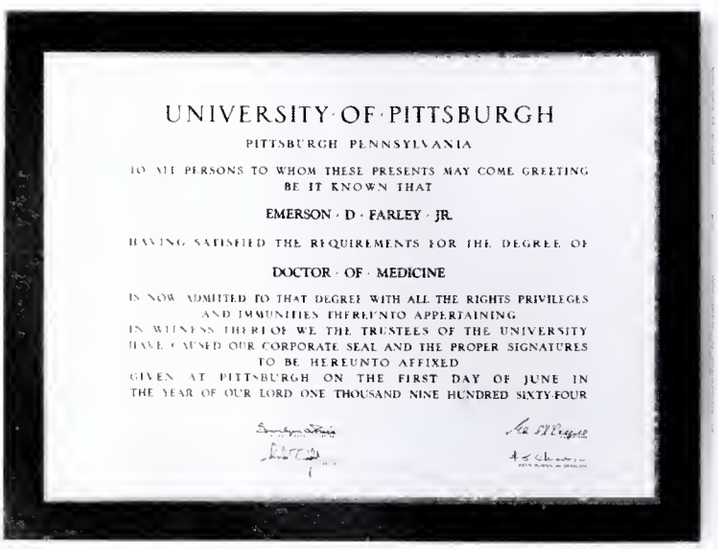
President

William D. Mabry Dean of School

Chairman, Board of Visitors

Secretary, Board of Visitors

Chairman: Ronald K. Davis, M.D., Richmond



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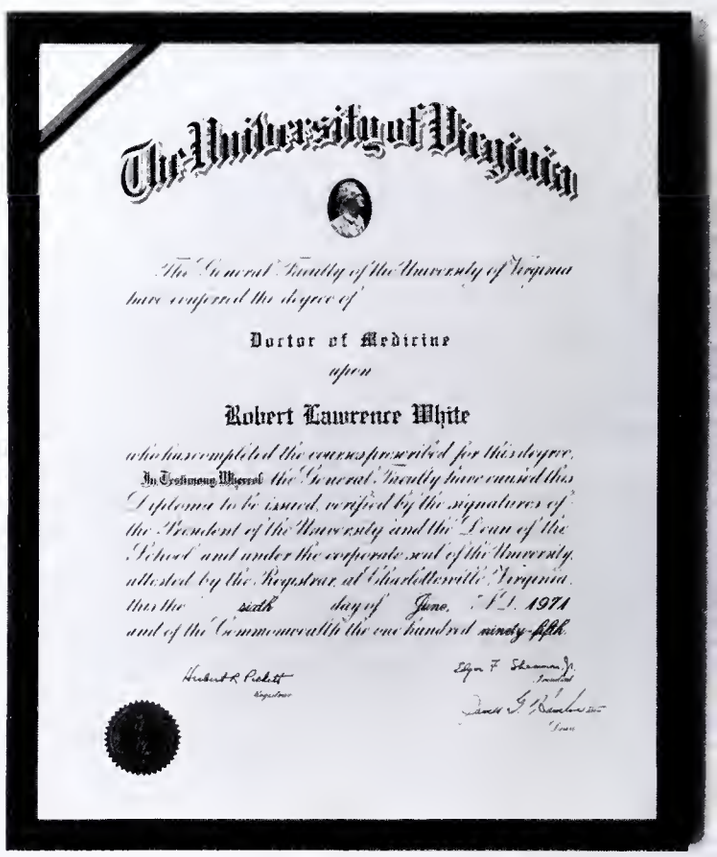

The University of Virginia

The General Faculty of the University of Virginia have conferred the degree of

Doctor of Medicine

upon

Robert Lawrence White

who has completed the courses prescribed for this degree.

In Testimony Whereof the General Faculty have caused this Diploma to be issued verified by the signatures of the President of the University and the Dean of the School and under the corporate seal of the University attested by the Registrar at Charlottesville Virginia this the sixth day of June A.D. 1971 and of the Commonwealth the one hundred ninety-fifth

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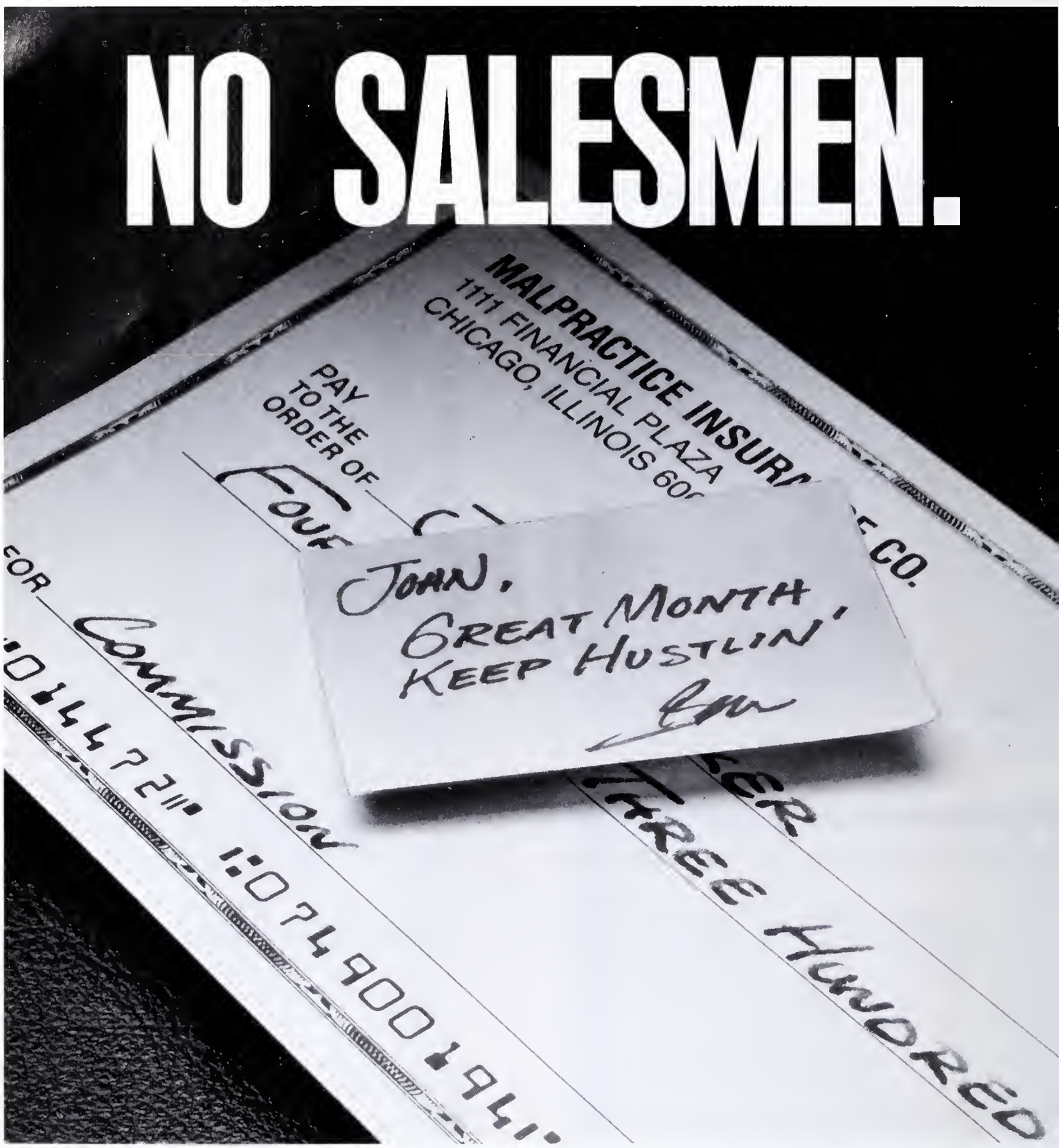

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Mutual Fears, Mutual Relief

What goes around, comes around. Forty years ago Walter Reuther, speaking for organized labor, was driving the AMA crazy with his call for national health insurance. (Instead, we got Medicare and Medicaid). Yet on February 17, 1991, at the Leadership Conference of the AMA, who was the invited speaker, the guest of honor? Lane Kirkland, president of the AFL-CIO—who else?

The circular system of history is always fun to analyze. Medicare and Medicaid got the taxpayers' attention, and a growing commitment to holding down health care costs, and as these rose governmental micromanagement became the order of the day—and still is. So costs were shifted to group health insurance, until employers began to scream. Then costs were shifted to individuals, non-workers and workers alike, and now they scream. Labor, willy-nilly, has to go to bat for workers vs. government management and the insurance industry. To build a coalition, labor is now reaching out to all the uncommitted players—the National Association of Manufacturers, the American Association of Retired Persons, and—yes, even to the American Medical Association.

This is not unwelcome; AMA President Tupper actually set the stage last fall, when he appeared before an AFL-CIO panel in San Francisco. The dialogue is important for both sides. Labor is trying to choose sides between two contrasting approaches: a monolithic plan like Canada's or a private non-profit system regulated by government, more like the Pepper Commission regulations or the AMA's Health Access America proposals. So far, they remain undecided.

Much as we physicians would like all these factions to simply shut up and go away, they won't. And before we hurry off to stick our heads in the sand, maybe we ought to listen to what patients are telling us.

What patients fear, more than anything else, is that illness will not only destroy their bodies and their lives but will insatiably devour their entire fortunes, leaving their families destitute even before death comes. The fear that they will run out of money before they run out of life is not irrational—look at how long we've been using the phrase "spend-down" to indicate how to get Medicaid support for nursing home care.

Complete helplessness in the face of progressive destruction produces the most terrible fear the soul can suffer. The only thing worse is to see everyone scampering away to avoid giving any meaningful help.

We physicians don't know fear like that—unless you've ever played a roulette "system" which requires you to bet always on red, and to double your bet whenever you lose. (Think how you feel when black comes up 17 times in a row, and you've gone from $1 to $131,072 per bet—with no end in sight.) We've suffered a milder version when malpractice insurance went skyrocketing—but that, in Virginia, is no longer the case. Mostly, we have a deep-seated fear of change. Nothing that HHS or HCFA or Medicaid or Blue Cross-Blue Shield can say will ever soothe our fears because all they ever do is change things—and change, as we see it, is always bad.

Both patients' and physicians' fears are realistic, but in our common predicament only the medical profession can take the lead. If we act to relieve our patient's deepest fears, at the cost of suppressing our own, the end result might be little short of miraculous. Overwhelming relief, and overwhelming gratitude, might well translate into overwhelming appreciation of the profession as a whole. That would be a change—but hardly the kind that makes us fearful. Because if the doctor-patient relationship grows ever stronger, we can take all other changes in our stride.

John A. Owen, Jr.

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Patient Self-Determination Act

New law ties living wills to hospitals, nursing homes, HMOs

Living wills are soon to become part and parcel of hospital admissions procedure in Virginia and all the other 49 states. This telling step was mandated by Congress when it quietly passed, last November, the Omnibus Budget Reconciliation Act of 1990 (PL 101-508). In §4206, this Act requires hospitals, skilled nursing facilities, hospices, home health agencies, and HMOs to provide each patient with 1) written information on the existing state law on living wills and durable powers of attorney for health care, and 2) their written policies respecting the implementation of these instruments. The new law also requires that all adult patients of these entities be asked whether they have executed such a directive. The provider must document the patient's treatment wishes and, if unable to implement those wishes, must transfer the patient to another facility.

Further, the Secretary of Health and Human Services is ordered to conduct a study to determine how the Act is being complied with, investigate methods of implementing patients' wishes when they are transferred to a new provider, and make recommendations for further legislation to carry out the purposes of the Act.

The provision becomes effective January 1, 1992. The penalty for noncompliance? Loss of Medicare and Medicaid reimbursement.

In Virginia both of the specified advance directives have been legalized: the Natural Death Act and its living will form were created by Virginia's General Assembly in 1984, and the durable power of attorney for health care was enabled by the Surrogate Decision-Making Statute of 1989. VMQ has assembled patient forms and physician guidelines for both Natural Death Act and durable power of attorney. All you have to do is fill out the coupon and put it in the mail. They are free of charge.

Both forms may be photocopied at will to give to your patients. Both can be completed without the assistance of an attorney, although they should be executed with all due care. Both are cognizant of Virginia law; they may not be viable in another state. Both require witnessing by two persons who are neither spouse nor blood relative of the signator. The durable power document asks for a notary seal; most banks provide notarization to their customers free of charge.

The original instrument should be kept in an accessible place—not a safety deposit box. The signator should also carry a wallet card indicating the existence of a surrogate decision-making instrument. Copies should be given to 1) close relatives of the signator; 2) the signator's treating physician; and 3) any person designated to make surrogate decisions.

The Natural Death Act form is valid until revoked, and unless limited to a specific time period by the document itself, so too are the durable powers. Either form can be revoked, but attorneys suggest that the revocation be in writing, in the manner of the original document, and that it be notarized. If the signator wants to designate a different surrogate or specify different treatment choices, the original document should be revoked and a new instrument written, signed, witnessed and notarized.

It is incumbent upon the treating physician to retain the document in the patient's medical record. Indeed, many physicians have begun routinely educating their patients to these documents and encouraging their use. Under the new law, such action will prove helpful to all concerned when a hospital admission is ordered. —A.G.

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National Data Bank Manual

Some indigestibles from a book that's "enough to make you sick"

When the editors asked me to review the *National Practitioner Data Bank Guidebook*,[1] I wondered if this was akin to asking a film critic to review one of the old Army training films. Actually, this manual is extremely well-written. So is Mary Shelley's *Frankenstein*.

The original draft of my earlier article on the Data Bank[2] began by calling it "vile and disgusting." The editors thought that a little strong and substituted "monstrous." I submit, however, that had they read the *Guidebook*, "vile and disgusting" would have remained. This thing is enough to make you sick. Some indigestibles:

1. When a malpractice plaintiff is paid money, and part of that money consists of the physician's contribution of the policy deductible, the physician must report him or herself to the Data Bank or face a $10,000 fine. (The insurance company also reports.)

2. If a patient makes a written demand for damages for malpractice and money is paid, it's reportable; if the demand is oral, it's not. (?!) If a bill for treatment is forgiven as settlement of a malpractice claim, it is not reportable.

3. A payment made by an insurer after creation of the Data Bank as part of a structured settlement entered into years ago is reportable, even if that settlement was 20 years ago.

4. Malpractice insurers cannot request information from the Data Bank, they must only provide it.

5. If a claim is against a physician and his/her group, if the group settles and the physician is dismissed, the claim need not be reported to the Data Bank unless the claim is against the P.C. of a sole practitioner. At least that's what I think it says on page 42. Check with your insurer to make sure.

6. Are you in a group practice which has a formal peer review system to ensure quality? If so, your group is a "health care entity" required to report to the Data Bank. This presumably would cover most HMOs, as the *Guidebook* points out. These groups must report certain disciplinary actions and may even inquire of the Data Bank for information about you before you sign up.

7. There is a full explanation for how to dispute the accuracy of a report filed about you. Essentially, when the Data Bank receives goodies about you, it is required to send an acknowledgment form to the reporting group and a copy to you. The Data Bank allows 60 days to straighten out inaccuracies.

8. While the Data Bank will tell you whenever you are reported, it will not tell you contemporaneously who has requested information about you. If you are in the Data Bank and request to see your file, the information provided will tell you of all queries to date. If you have never received a copy of a report acknowledgment, then you are not in the Data Bank files.

9. If a malpractice plaintiff sues a hospital alleging improper staff credentialing and a part of that claim is an allegation that the Data Bank was not queried by the hospital, your file can be obtained, presumably to show that had the hospital made the inquiry, it would not have given you privileges. Or something like that.

And so on. This book is intended to tell you anything you could possibly want to know about the Data Bank, and how your name gets into it. What it amounts to, however, is a well-organized, systematic description of the raping of the civil liberties of an oppressed class: health care providers. Are you really paid enough to make you put up with this intrusion into your professional and personal privacy, to suffer this wholly unjustified denial of equal protection under the law?

Maybe someone has bought the movie rights to this book. Why not? There are already a slew of successful movies about Big Brother and other government-type monsters. This fits right in.

—JOHN D. GRAD

1. National Practitioner Data Bank Guidebook, sponsored by the U.S. Department of Health and Human Services. McLean, Virginia, Unisys Corporation System Development Group, 1990, 80 pp.
2. Grad JD. Will National Data Bank encourage litigation? VMQ 1990;Autumn(Oct):343-4

John Grad is counsel to the Alexandria Medical Society and a partner in the law firm of Grad & Logan, 112 North Columbus Street, Alexandria VA 22313.

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WHO'S WHO

Dr. Read F. McGehee, Jr., Richmond, has been elected president of the Virginia Board of Medicine, succeeding **Dr. F. J. Pepper**, Alexandria, at the helm of the 16-member regulatory body. Elected secretary-treasurer on the same slate was **Dr. Charles F. Lovell**, Norfolk. They will take over their duties on June 6.

A native of Sumter, South Carolina, Dr. McGehee came to Virginia to earn his MD at the Medical College of Virginia (M-1961). He is a pulmonary and infectious diseases specialist, and early in his career was an epidemiologist at the Communicable Disease Center in Atlanta. A former president of the medical staff of St. Mary's Hospital, Richmond, he is now chairman of that hospital's Medical Executive Committee.

The State Emergency Medical Services have a new medical director in **Dr. Gaylord W. Ray**, Gloucester, who has been a member of the Governor's EMS Advisory Board since 1984 and has served as chief medical advisor for the Virginia Association of Volunteer Rescue Squads since 1987. Named Outstanding Operational Medical Director for 1990 by the State EMS was **Dr. Donald A. Sabella**, Loudoun.

Dr. Philip T. Rodilosso, Arlington internist and a past president of the Virginia Society of Internal Medicine, is the new president of the Medical Society of Virginia Review Organization. A member of the MSVRO board since 1984, he is a graduate of Georgetown University School of Medicine, where he is now a clinical associate professor of medicine.

Every now and then someone wanders into the waiting area of **Dr. Richard H. Fisher**'s office in Salem and asks to buy some stamps. This is not surprising, because Dr. Fisher's office is in the Old Salem Post Office building. When the post office moved to its new facility down the street, the orthopedist approached city officials with a bid for the old building, and the deal was soon done. From the first Dr. Fisher knew what he wanted to do with the building: move his practice into it.

The Fisher family has a predilection for activities postal. Dr. Fisher's father was in mail travel service, his brother Robert was a postal inspector, and his younger brother Harold worked for the post office while in college. Dr. Fisher himself worked on a train sorting mail over Christmas during medical school to get a microscope, he told reporter Terry Monaghan of the *Salem Times-Register*. And as a lifelong Salem resident, "I have always taken pride in the building," he added.

That pride is expressed in the renovations that took 15 months to complete. When the doors finally opened, early this year, three tenants signed up to share the building. Dr. Fisher's space includes an x-ray room, surgery and examining rooms.

In recognition of his many years of service as clinician, educator, and leader in dermatology, **Dr. Peyton E. Weary,** Charlottesville, has received from the American Academy of Dermatology its highest award, the gold medal. Only ten gold medals have been presented by the academy in the past 28 years. Dr. Weary has been at the University of Virginia since enrolling in the School of Medicine in 1951. He is currently chairman and professor of dermatology.

Dr. Robert Dea Ailsworth, Keysville, had announced to his patients that he was going to retire from family practice on 1 January 1991, but when no replacement for him could be found, he agreed to postpone his leavetaking, and to the great relief of his patients, he was still plugging away as this was writ-

Dr. Fisher: orthopedics among the mail boxes

Salem Times-Register photo by Terry Monaghan

ten. A graduate of William and Mary College and the Medical College of Virginia, Dr. Ailsworth has been serving the residents of Charlotte and Prince Edward Counties since 1951.

At the Eye Bank Association of America's meeting in Atlanta, **Dr. Walter Mayer**, Richmond ophthalmologist, was honored with the R. Townley Paton MD Award for outstanding contributions to eye banking. Dr. Mayer recently retired after 20 years as medical director of the Old Dominion Eye Bank, Richmond. He is a founding member and past president of the EBAA.

Virginia's Southern Baptists, at a convention in Richmond, elected as their president **Dr. Michael J. Oblinger,** Charlottesville gastroenterologist. There are 1,300 Southern Baptist churches in Virginia with an estimated 600,000 members.

Medical director of the new Greensville Correctional Center in Jarratt is **Dr. John A. Holland, Jr.,** family physician with a private practice in Emporia. Dr. Holland is a graduate of Meherrin Medical College and did his FP residency at Norfolk General.

"Sorely missed by the townspeople" of Scottsville, according to a report in Charlottesville's *Daily Progress,* is **Dr. William E. Moody**, who retired late in 1990 after 40 years of general practice. Not only was Dr. Moody "an exceptionally good doctor," Mayor A. Raymon Thacker told a reporter, but his retirement means that Scottsville no longer has a full-time physician. For Moody, the retirement was "the hardest job I ever had" but was necessitated by age.

Born in Cleveland, Ohio, in 1908, Dr. Moody was graduated from the medical school at Western Reserve University and arrived in Scottsville in 1946 after serving as a U.S. Army physician in the South Pacific during World War II. Every year he and his wife spent two weeks in Florida; for the other 50 weeks he was available around the clock to the sick and injured of Scottsville and the surrounding area.

Two members of the Medical Society of Virginia were elected to fellowship by the American College of Radiology at its fall meeting in Nashville, Tennessee. They are **Dr. John A. Long, Jr.**, of Bethesda, Maryland, and **Dr. Howard F. Faunce** of Richmond.

Gov. L. Douglas Wilder has appointed **Dr. M. Pinson Neal, Jr.**, Richmond, to the State Board of Forestry.

Dr. Richard P. Keeling, director of student health at the University of Virginia, Charlottesville, has received the American College Health Association's highest award, the Edward Hitchcock Award, for extraordinary contributions to the field of college health, especially for his work in the fight against HIV disease. Dr. Keeling is a past president of the Association.

The Southern Medical Association's new councilor for Virginia is **Dr. Robert W. Klink**, Gloucester ob/gyn, who was chosen during the SMA's annual scientific assembly in Nashville.

A family physician, **Dr. Richard C. Cole,** Stuart, was named one of six Outstanding Young Virginians for 1991 by the Virginia Jaycees. A lifelong resident of Patrick County, Dr. Cole practices medicine, pediatrics and obstetrics at the Stuart Clinic and is active in the youth programs of the 4-H and Stuart Rotary Clubs. He is a graduate of the University of Virginia School of Medicine.

An endowed nursing scholarship has been established at Patrick Henry Community College, Martinsville, in the name of retired Martinsville physician **Dr. Jethro Hurt Irby.** The fund was enabled by a donation from a Martinsville resident and will yield full-time scholarships for two nursing students each year, including tuition, books and fees.

Dr. Irby was a family practitioner in Martinsville from 1946 until his retirement in 1988; he was active in church and community organizations and could always be counted on to support local efforts on behalf of education. He is an alumnus of the University of Virginia School of Medicine.

At the American Society of Plastic and Reconstructive Surgeons' recent scientific meeting in Norfolk, **Dr. Charles E. Horton**, Norfolk, was tapped for the 1990 ASPRS Achievement Award. Chairman of the Department of Plastic Surgery at Eastern Virginia Medical School, Dr. Horton founded and is president of Physicians for Peace, which sponsors teams of volunteer doctors and nurses who care for the medically needy in the Middle East.

Dr. W. Allen Fuller, Jr., of South Boston, was initiated as a fellow of the American College of Surgeons at its recent clinical congress in San Francisco.

Dr. Beverly J. Loesch, Waynesboro, has retired from her practice of internal medicine but still serves for other area physicians when needed, according to a story in the Waynesboro *News-Virginian*, and is also into a heavy schedule of volunteerism: president of Hospice, member of the Commission on the Elderly and the Valley Health Council, and on the Adult Day Care and Community Concert boards. She's on the road a lot, too, to keep in touch with her four children and six grandchildren, who are scattered from Charlottesville to New Jersey.

Dr. Calvin L. Miller, Abingdon, is the new chief of staff at Abingdon's Johnston Memorial Hospital. He is an ophthalmologist.

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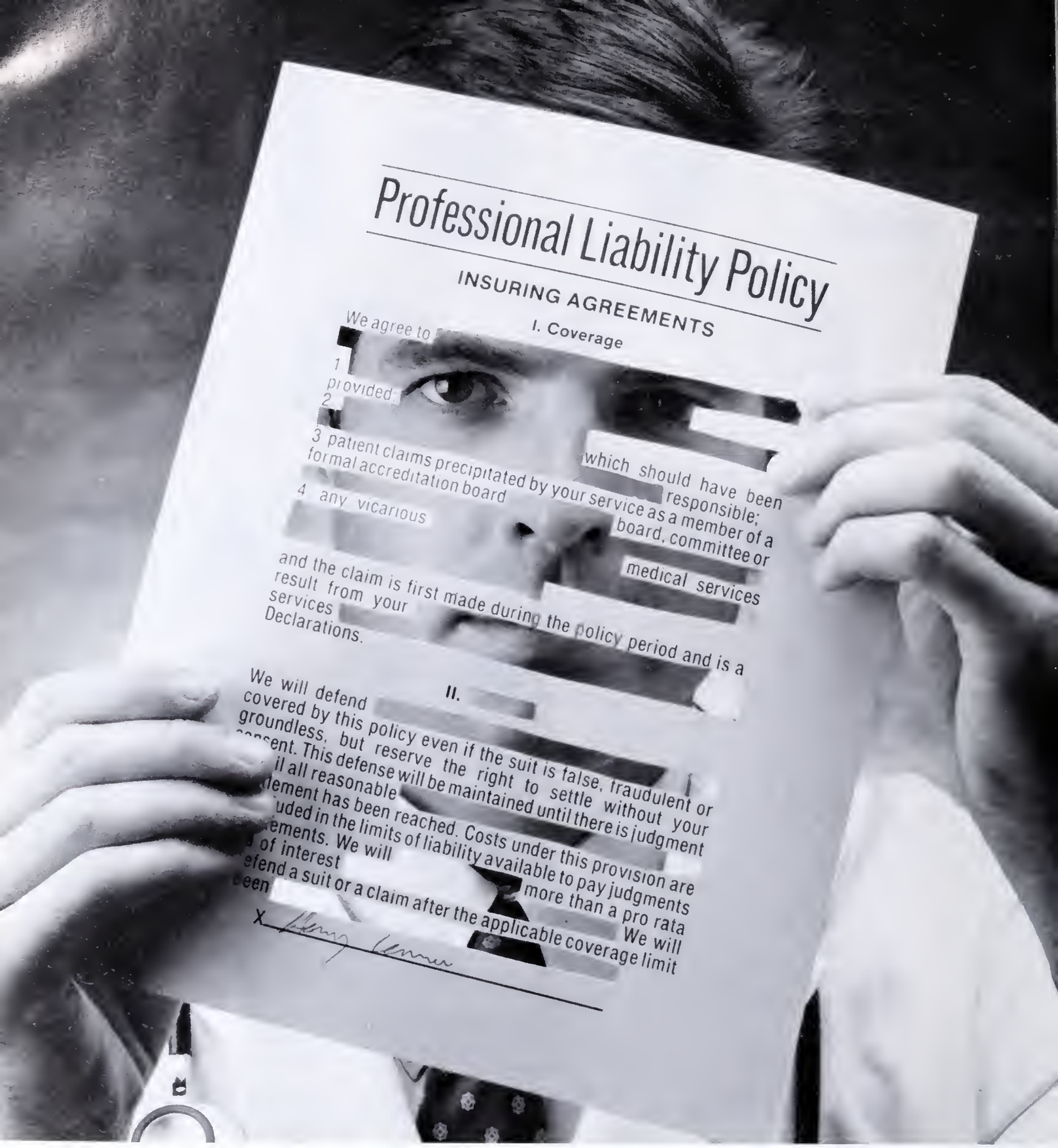

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MEETINGS

**1991 Annual Meeting,
The Medical Society of Virginia,
November 6-10, Williamsburg**

April 20
Regular meeting of the Medical Society of Virginia's Council, *Richmond*. James L. Moore, Jr., 804-353-2721.

April 22-24
McLemore Birdsong Pediatric Conference (University of Virginia), *Charlottesville*. 18 credit hrs. Fee: $330. Pam Breeden, 804-924-9148.

April 25
Substance Abuse in Maternal and Child Health (Virginia Department of Health/Eastern Region), *Virginia Beach*. 6 credit hrs. Fee: $40. Marjorie C. Ruegger, 804-363-3874.

April 25-27
Advances in Hip and Knee Arthroplasty (Johns Hopkins), *Williamsburg*. 18 credit hrs. Fee: $575. CME Office, 301-955-2959.

April 26-28
26th Annual Pediatric Springfest (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

May 3-5
Annual Meeting of the Virginia Surgical Society, *Williamsburg*. Irving L. Kron, MD, 804-924-2158.

May 3-5
Diagnostic Dilemmas in Neurology and Psychiatry, (Southern Medical Association), *Point Clear, Alabama*. 15 credit hrs. Fee: $325. 205-945-1840.

May 9-12
Annual Meeting of the Virginia Chapter, American College of Radiology, *Williamsburg*. Patricia R. Berry, 703-669-8312.

May 10-11
Annual Meeting of the Virginia Society of Ophthalmology, Hyatt House, *Reston*. Donna Scott, 804-353-2721.

May 15-19
3rd Baltimore Perinatal Colloquium (Johns Hopkins), *Baltimore*. 24 credit hrs. Fee: $450. CME Office, 301-955-2959.

May 16-17
Pediatric Allergy/Immunology for the Practitioner (Johns Hopkins), *Baltimore*. Fee: $195. CME Office, 301-955-2959.

May 17-18
Annual Meeting of the Virginia Society of Otolaryngology/ Head and Neck Surgery, Omni Hotel, *Norfolk*. Donna Scott, 804-353-2721.

June 22, 1991

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May 25-27
12th Annual Conference on Urogynecology and Pelvic Surgery (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

June 2-9
10th Annual Reproductive Endocrinology and Gynecology Seminar (Johns Hopkins), *Hilton Head, South Carolina*. Fee: $850. CME Office, 301-955-2959.

June 3-7
7th EVMS Family Medicine Review Course (Eastern Virginia Medical School), *Virginia Beach*. 42 credit hrs. Fee: $450. CME Office, 804-446-6140.

June 3-14
4th Summer Institute in Environmental Health Studies (Johns Hopkins), *Baltimore*. Dr. Jacqueline Corn, 301-955-2609.

June 6-9
15th Annual Postgraduate Course on Rehabilitation of the Brain-Injured Adult and Child (Medical College of Virginia/VCU), *Williamsburg*. CME Office, 804-786-0494.

June 9-13
15th Symposium on Lung Disease, *Sea Island, Georgia*. 24 credit hrs. Fee: $475. Southern Medical Association, 205-945-1840.

Continued on next page

MORE MEETINGS

June 13-15
36th Great Smoky Mountains Pediatric Seminar (University of Tennessee), *Gatlinburg, Tennessee*. CME Office, 615-544-9190.

June 14
Cardiopulmonary Stress Testing and Indirect Calorimetry (Eastern Virginia Medical School), *Virginia Beach*. Jeanette Schmitz, 804-446-6143.

June 14-16
Annual Meeting of the Virginia Allergy Society, Ramada Oceanfront Inn, *Virginia Beach*. Donna Scott, 804-353-2721.

June 17-19
Workshop Conference on Breast Cancer Diagnosis: Interventional Procedures (Washington University), *Hilton Head, South Carolina*. Siemens Medical Systems, 414-784-1455.

June 19-23
18th Annual Art and Science of Sports Medicine Conference (University of Virginia), *Charlottesville*. 3 credit hrs. Dr. Joe Gieck, 804-982-5450.

June 19-23
1st Annual Spring Meeting of the Southern Association for Geriatric Medicine, *Vancouver, British Columbia*. Robin Buchanan, 205-945-8425.

June 20-23
15th Edition, Practical Dermatology for the Primary Care Physician (Eastern Virginia Medical School), *San Francisco, California*. 22 credit hrs. Fee: $450. CME Office, 804-446-6140.

June 22
11th Annual Advances in Gastroenterology (Presbyterian University of Pennsylvania/Underwood Memorial Hospital), *Atlantic City, New Jersey*. Registration Manager, 609-848-1000.

June 28-30
9th Summer Symposium in Internal Medicine (Eastern Virginia Medical School), *Williamsburg*. 12 credit hrs. CME Office, 804-446-6140.

June 28-30
Diagnosis and Management of Respiratory Diseases, *Williamsburg*. Medical Education Resources, 1-800-421-3756.

July 10-14
14th Annual Flap Dissection Workshop (Eastern Virginia Medical School), *Virginia Beach*. Jeanette Schmitz, 804-446-6143.

July 12-14
Heart Failure, Transplantation, and Interventional Cardiology (Medical College of Virginia), *Virginia Beach*. CME Office, 804-786-0494.

July 19-21
13th Annual Pediatric Primary Care Conference: Pediatrics at the Beach (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

July 26-28
Practical Internal Medicine Conference (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

August 2-4
8th Annual National Clinical Care Diabetes Conference: Diabetic Neuropathy (Eastern Virginia Medical School), *Virginia Beach*. Jeanette Schmitz, 804-446-6143.

August 5-9
1991 Summer Symposium: Trauma, an Emergency Physician's Perspective (Virginia Chapter. American College of Emergency Physicians), *Virginia Beach*. 27 credit hrs. Gwen E. Harry, 804-966-5966.

August 8-11
15th Annual Summer Retreat: Practical Issues in Primary Care (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

August 15-18
Plastic Surgery of the Eyelids and Orbit/Oculoplastic Surgery: Aesthetic and Reconstructive (Eastern Virginia Medical School), *Virginia Beach*, Jeanette Schmitz, 804-446-6143.

August 16-18
7th Annual Conference on Primary Care of the Female Patient (Medical College of Virginia/VCU), *Virginia Beach*. CME Office, 804-786-0494.

September 14
Regular meeting of the Medical Society of Virginia's Council. *Richmond*. James L. Moore, Jr., 804-353-2721.

September 19-21
Virginia Occupational Health Conference (Eastern Virginia Medical School), *Williamsburg*. Jeanette Schmitz, 804-446-6143.

September 20
14th Annual Perinatal Conference (Medical College of Virginia), *Richmond*. CME Office, 804-786-0494.

September 21-23
Annual Meeting of the Virginia Vascular Society, Tides Lodge, *Irvington*. Dr. Walter H. Graham, 804-596-7631.

October 12-13
Pediatric Advanced Life Support Provider Course (Mary Washington Hospital), *Fredericksburg*. Limited to 24 students. Dr. Melville G. Wright III, 703-899-1326.

December 5-7
Advances in Obstetrics and Gynecology (Medical College of Virginia), *Richmond*. CME Office, 804-786-0494.

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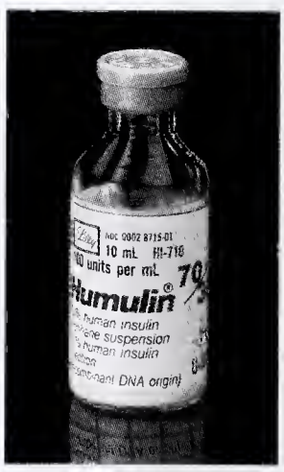

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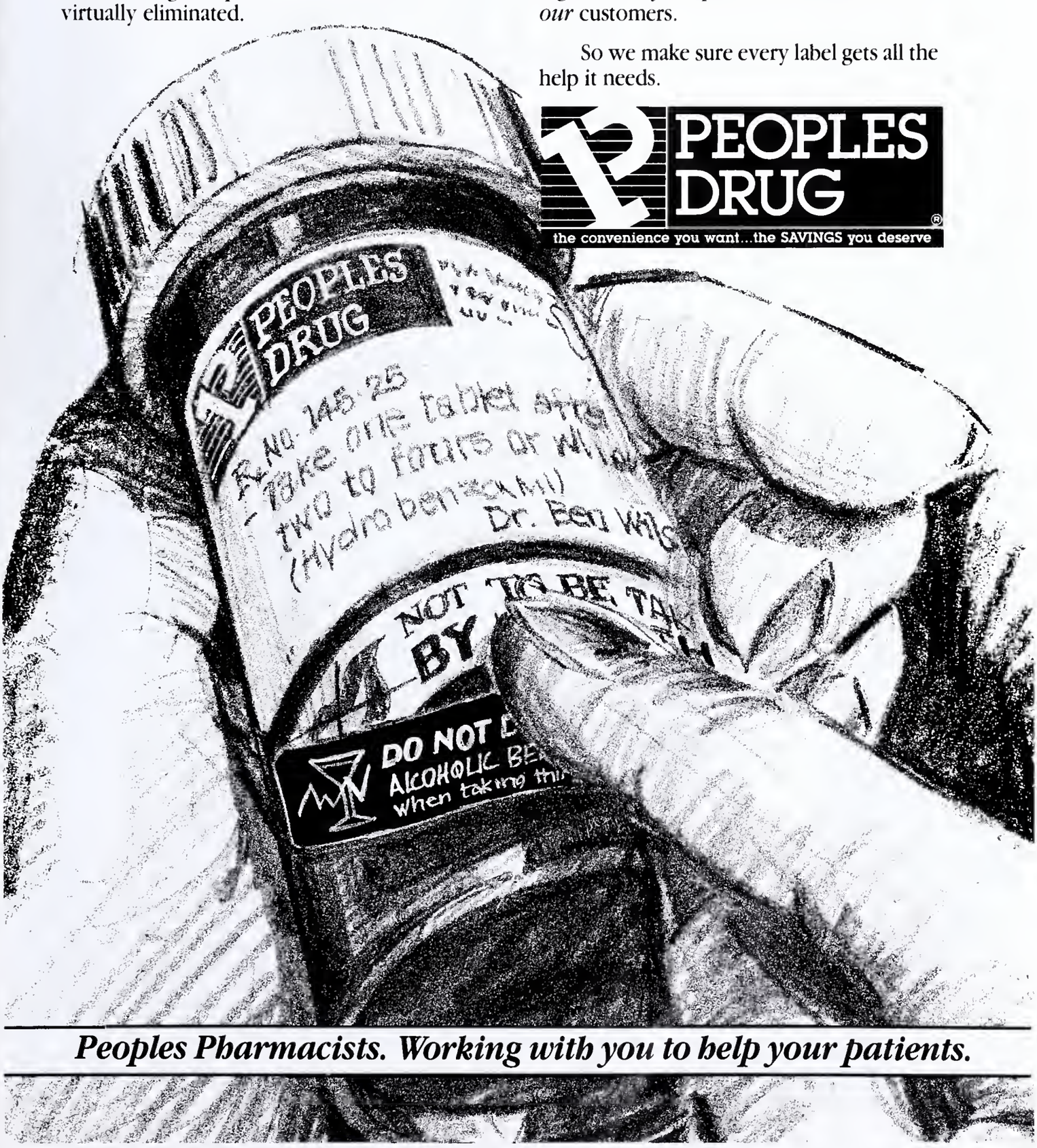

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CARING FOR THE AGED

THE six articles in this section derive from the Second Annual Conference on Aging sponsored by the Division of Geriatric Medicine, Medical College of Virginia/Virginia Commonwealth University, and McGuire Veterans Affairs Medical Center on December 6-9, 1990, in Williamsburg. Thomas Mulligan, MD, and Michael Godschalk, MD, were program directors. The topics were selected for their relevance to the primary care physician's complicated task of caring for elderly patients.

An area of special significance is functional status assessment. In their article on geriatric dysfunction, Mulligan and Teitelman deal with one aspect of functional status often overlooked by practitioners, male sexual dysfunction.

Despite the public enthusiasm for health promotion, most of the focus has been on the non-elderly. Two articles in this section deal with health maintenance for the elderly: Godschalk's text on the treatment of hypercholesterolemia and a second entry from Mulligan and Teitelman, this one on preventive gerontology.

Two topics from the Conference remind us that in the elderly we find the highest prevalence of chronic illness. Schmitt reviews current concepts in the management of congestive heart failure. Scott provides a broad perspective on the epidemiology and prevention of cancer in the elderly.

Finally, McMurtry deals with ethical dilemmas in geriatric care, presenting two case reports that illustrate the need for physicians to play an active role in eliciting from elderly patients advance directives for their care.

Elderly patients present the primary care physician with a difficult task: How to balance efficiently the delicate tasks of providing enough care while avoiding iatrogenic harm. When in doubt the geriatric maxim should apply: "Start low and go slow."

SHELDON M. RETCHIN, MD

Dr. Retchin is chairman of the Division of Geriatric Medicine, Department of Internal Medicine, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to him at Box 287, MCV Station, Richmond VA 23298.

VMQ
SPRING 1991

Geriatric Sexual Dysfunction

Thomas Mulligan, MD, and Jodi Teitelman, PhD,
Richmond, Virginia

More than half of males and females over the age of 80 report continued interest in sex but declining intercourse frequency, with a high incidence of male impotence. The authors cite nine common causes of erectile failure in the aged. Available treatment modalities include pharmacotherapy, external suction devices, introcorporeal injections, and penile prostheses.

THE FREQUENCY of sexual activity declines dramatically from young adulthood to old age. Fewer than 15% of men or women over the age of 80 engage in intercourse.[1] In contrast, sexual interest (libido) and orgasmic ability decline to a much lesser degree. Greater than 50% of males or females over the age of 80 report continued interest in sex.[2] Declining intercourse frequency, despite continued interest, is due to multiple factors, but most frequently inability of the male to perform: impotence. Unfortunately, this "libido-potency gap" widens with advancing age.[3] Because impotence is the most common geriatric sexual dysfunction, we will focus our attention on this common clinical problem.

Normal Sexual Physiology

Testosterone and its metabolites function primarily in embryonic differentiation and maintenance of genital and central nervous system target tissues. Furthermore, testosterone appears to mediate sexual motivation. The role of testosterone in facilitating erections has yet to be proven.

The stimulus for erection can be either psychogenic or local. Psychogenic stimuli traverse the limbic system and autonomic nervous system. Sympathetic activity is probably responsible for tonic constriction of the cavernosal arterioles and sinusoidal spaces, whereas parasympathetic activity is responsible for penile arteriolar dilation.[4]

Local stimulation of free nerve endings in the glans penis and surrounding skin, as well as deeper stimulation of Pacinian corpuscles produce impulses which traverse the somatic branches of the pudendal nerve to the sacral spinal cord (S_{1-2}).[5] From the sacral cord, nerve impulses return to the penis via the parasympathetic fibers of the Nervi Erigentes (Pelvic Nerve).

Neurotransmitters found within the corpora cavernosa include acetylcholine, norepinephrine and vasoactive intestinal polypeptide (VIP). VIP levels increase in the penile vein during erection. Although the details of the neurotransmitter interactions await further investigation, erection is likely dependent upon acetylcholine and VIP. Adrenergic neurotransmitters, on the other hand, may be related to detumescence.

With release of neurotransmitters, penile rigidity develops through a marked increase in penile arterial

From the Division of Geriatric Medicine, Medical College of Virginia/Virginia Commonwealth University and McGuire Veterans Affairs Medical Center (Dr. Mulligan), and the Department of Gerontology, MCV/VCU (Dr. Teitelman). Address correspondence to Dr. Mulligan at the McGuire Veterans Affairs Medical Center (181), 1201 Broad Rock Road, Richmond VA 23249.

inflow.[6] Initial tumescence occurs through dilation of intracorporeal sinusoids, decreased intracorporeal resistance and dilated cavernosal arterioles.[7] As intracorporeal sinusoids dilate, the subalbugineal venular plexuses are compressed and venous outflow is restricted.[8] Further arterial inflow, with relative outflow obstruction, results in rising pressure within the non-elastic tunica albuginea and penile rigidity.[9]

The neural pathways of ejaculation are similar to those for erection, with the exception that the sympathetic system plays the major role. Impulses from the limbic system traverse the sympathetic chain enroute to the T_{12}-L_1 cord, and then to the penis via the hypogastric nerves. Sympathetic activity causes rhythmic contractions of the vas deferens, prostate and seminal vesicles, thereby giving rise to ejaculation.[10] The emotive aspects of orgasm are likely related to systemic release of catecholamines, but the specific details remain unclear.

Prevention of Erectile Failure

To prevent age-associated erectile failure, one must first understand its causes. Erectile rigidity requires a complex interaction of the psychologic, neurologic and vascular systems. Common aspects of "normal" aging, such as slowed nerve conduction velocity, result in the need for greater stimulation than was required during the younger years to achieve an erection but not frank erectile failure. When age-associated diseases, such as diabetes or atherosclerosis, are superimposed on the normal changes of aging, erectile failure becomes prevalent. The most common causes of erectile failure in the aged include:[11]

Multifactorial (neuro-vascular)	30.3%
Vascular	21.1%
Diabetic neuropathy	17.1%
Non-diabetic neuropathy	10.5%
Psychogenic	9.2%
Drug effects	3.9%
Hypogonadism	2.6%
Peyronie's disease	1.3%
Idiopathic	3.9%

In view of the common causes of impotence in older men (e.g., neurovascular disease) and the known prevalence of atherosclerosis and diabetes mellitus with aging, one can see that recommendations for a healthy lifestyle (e.g., exercise, diet) will not only help prevent heart disease and stroke, but also impotence. The factors that likely result in age-associated erectile failure include sedentary lifestyle, high intake of cholesterol and concentrated carbohydrates, as well as tobacco, alcohol and drug abuse.

However, despite our best efforts at prevention, clinicians will probably always be faced with aging men complaining of impotence. In this case, knowledge of the therapeutic options will make rational treatment feasible.

Treatment of Erectile Failure

Available treatment modalities include elimination of adverse drug effects, pharmacotherapy, sex therapy, external suction devices, intracorporeal injections, vascular surgery, and penile prostheses.[12] Smoking cessation should be encouraged to provide even marginal improvement in penile arterial flow.

Patients with hypogonadism will usually report impaired libido rather than simply impaired erections. Nevertheless, in patients with hypogonadism, testosterone, 200 mgIM every three weeks, is effective.[13] Oral testosterone undecanoate is also effective, but may be associated with cholestasis. Yohimbine, an alpha-2 receptor antagonist, appears to increase libido but has no proven effect on erectile rigidity.

Of the external devices currently available, one utilizes vacuum induction to produce an erection (Erec-Aid®, Osbon). The initial study reported a good response rate and few adverse effects other than occasional ecchymoses;[14] however, this device requires a degree of manual dexterity which may preclude its use by elderly males with arthritic hands. Another external device (Erection System®, Synergist) utilizes suction to draw the penis into a silastic condom-shaped device, which remains on the penis during intercourse. The innate stiffness of this device, augmented by the tumescent penis, allows vaginal penetration. Both devices are most effective when used in a stable and supportive relationship.

Intracorporeal pharmacotherapy is currently the area of most active investigation. Autoinjection of papaverine, either alone or in combination with phentolamine, or prostaglandin E_1 will produce penile erections.[15] However, the risk of adverse reactions, such as priapism, warrants its use only by experienced urologists. Long-term studies on efficacy and safety are still in progress, with penile fibrosis after long-term use of papaverine being the greatest concern.

If other less invasive treatments have failed, or if the patient has penile arterial insufficiency, surgical implantation of a penile prosthesis may be the best remaining option. A variety of prostheses are available, but the multi-component inflatable device usually results in the most satisfactory outcome.

Summary

Most aged couples no longer engage in intercourse due to male sexual dysfunction: impotence. Prevention of impotence is quite similar to prevention of other vascular and neurologic diseases; a healthy lifestyle throughout the lifespan is the best way to prevent disease regardless of the organ involved. Nevertheless, even when prevention has not occurred, treatment is often successful because of the variety of options now available. With a compassionate and conscientious approach to the management of impotence in aged men, the sexual component to quality of life can often be regained.

Should We Treat High Cholesterol in the Aged?

Michael F. Godschalk, MD,
Richmond, Virginia

In the past few years cholesterol has become a high priority topic in both the medical and the lay press. However, the subject of high cholesterol in the aged has been controversial, primarily because there are no studies that specifically look at the elderly. The goals of this review are to examine the risk of hypercholesterolemia in the aged and to discuss the rational evaluation and treatment of this disorder.

Epidemiology

The National Cholesterol Education Program (NCEP) classifies a total blood cholesterol of less than 200 mg/dL as desirable, 200 to 239 mg/dL as borderline, and a total cholesterol of 240 mg/dL or greater as high.[1] The prevalence of high cholesterol increases with age, so that by age 75, approximately one-third of men and over one-half of women in this country have high cholesterol levels. Since there are approximately 30 million people over the age of 65 in the United States, between 10 and 15 million elderly have hypercholesterolemia. Thus it is important to establish whether hypercholesterolemia is still a significant risk factor in the elderly.

The Framingham Heart Study prospectively examined the risk of coronary heart disease (CHD) in 2,282 men and 2,845 women, aged 30 to 62 years.[2] The subjects were divided into groups based on cholesterol levels and were followed for 14 years. In this study, the relative risk of coronary heart disease appeared to decrease with increasing age. Similar results were seen in the Multiple Risk Factor Intervention Trial (MRFIT) in which the relationship between serum cholesterol and the six-year CHD mortality was studied in 356,222 men.[3] These data have been used to argue that high cholesterol is not an important risk factor in the elderly. This conclusion is not justified, because relative risk does not take into account the rising prevalence of CHD with age, nor does it recognize that cardiac disease is the most common cause of death in the elderly.

A better way to examine these data is to look at attributable risk. Attributable risk estimates how much coronary heart disease would be prevented if high cholesterol levels were reduced to normal levels. Using the MRFIT data, instead of decreasing with age, the attributable risk more than doubles from the lowest to the highest age group.[4] Therefore, although the relative risk of CHD associated with a high cholesterol level is greater in young subjects compared to the elderly, the absolute number of subjects benefiting from lowered high cholesterol levels would be greater in the elderly than in the young.

Evaluation

The NCEP recommends that patients with a total cholesterol level <200 mg/dL should be rechecked

From the Division of Geriatric Medicine, Medical College of Virginia/Virginia Commonwealth University, and the McGuire Veterans Affairs Medical Center. Address correspondence to Dr. Godschalk at McGuire Veterans Affairs Medical Center (111S), 1201 Broad Rock Road, Richmond VA 23249.

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every five years. For those in the borderline group, 200-239 mg/dL, need for treatment is based on the presence or absence of risk factors: male sex, smoking, hypertension, diabetes, severe obesity and a history of CHD. Patients without CHD or two of the other risk factors should be rechecked yearly. If CHD or two of the risk factors are present, or if the cholesterol level is high, ≥240 mg/dL, a lipid profile should be obtained.

Risk factors, however, are frequently present in the elderly. Therefore, if the cholesterol level is ≥200 mg/dL, lipoprotein analysis is warranted in most cases. Before making a decision about treatment, two or more measurements need to be obtained, since the coefficient of variation for total and HDL cholesterol is about 5% and 14% respectively.[5] Lipoprotein analysis is expensive, with charges from $26 to $58 depending on where the test is done. In addition to repeating the measurements before treating a high cholesterol level, underlying diseases such as diabetes and hypothyroidism need to be treated appropriately. Also, drugs that affect lipid levels (β-blockers, thiazides, glucocorticoids, etc.) should be discontinued if possible.

Therapy

Although there are no studies focusing specifically on the elderly, there are studies that include older subjects. In a trial looking at the effect of diet on death due to CHD or stroke, Dayton et al reported that at the end of eight years of therapy, mortality was decreased in subjects with high cholesterols irrespective of age.[6] A second study, the Lipid Research Clinics Coronary Primary Prevention Trial, examined the effect of cholestyramine and diet on cholesterol levels and found that after seven years, older men had a greater reduction in total cholesterol and LDL cholesterol than did younger men.[7]

Based on these data, treatment, whether dietary or cholestyramine, seems to work in the elderly. However, it may take several years to see a reduction in CHD morbidity and mortality.

In general, diet therapy should always be considered first (Table 1). Dietary therapy is divided into two "steps." Step one reduces saturated fat intake to less than 10% of total calories and decreases dietary cholesterol intake to less than 300 mg/day. Step two further decreases saturated fat intake to less than 7% of total calories and cholesterol intake to less than 200 mg/day. These diets are not easy to follow, especially for an elder, who may not have the financial resources or be physically able to prepare food that is low in saturated fat and cholesterol. A dietitian is needed to instruct the patient and/or care giver in the diet. The step one diet should be tried for three months, and if there is not a significant decrease in LDL levels, the step two diet should be tried. If diet fails, drug therapy may be indicated.

Although scientific data are inadequate, nicotinic acid or one of the bile acid sequestrants may be the drugs of choice, because, at least in middle-age subjects, their efficacy and safety have been well established. Nicotinic acid is available without prescription and is, therefore, very inexpensive. It decreases the synthesis of lipoproteins by the liver, lowering LDL and triglycerides and raising HDL. Its main drawback is cutaneous flushing, which can be avoided by "starting low and going slow." We start with 50 mg three times a day with meals for one week and then increase the dose by 50 mg each week. Starting with a low dose usually avoids flushing. Since flushing, if it does occur, is prostaglandin mediated, it usually can be prevented by taking one aspirin 15 to 30 minutes before each dose of nicotinic acid.

Table 1. National Cholesterol Education Program Treatment Recommendations.

LDL (mg/dL)	Risk Factors	Therapy
<130		Desirable
130-159	Absent	Recheck yearly
	Present	Begin diet therapy
≥160	Absent	Begin diet therapy
	Present	Begin drug therapy
≥190		Begin drug therapy

The bile acid sequestrants decrease resorption of bile acids in the gut, increasing the hepatic clearance of LDL. They are very effective at decreasing LDL, but may increase triglycerides and should not be used in patients with hypertriglyceridemia. Their main side effect is constipation, which can be avoided by starting with a low dose, increasing dietary fiber and oral fluid intake. We usually start with 4 to 5 grams twice a day, and if LDL levels are not adequately decreased after several weeks, gradually increase the dose.

Gemfibrozil raises HDL and lowers both LDL and triglyceride levels. It is usually given in a dose of 600 mg bid. Side effects include cholesterol gallstones and myopathy. Because of these potential side effects, we limit the use of gemfibrozil to patients with hypercholesterolemia and hypertriglyceridemia.

Because of its expense, lovastatin may be best reserved for patients whose LDL levels cannot be adequately controlled with either nicotinic acid or bile acid sequestrants. Lovastatin blocks the synthesis of cholesterol by the liver and is very effective in decreasing LDL cholesterol. When necessary, we start with a dose of 10 mg/day. Lovastatin may cause an elevation in liver enzymes. It may also cause myopathy and, rarely, myoglobinuria with resulting acute renal failure. Because lovastatin and gemfibrozil may both cause myopathy and possibly rhabdomyolysis, they should not be given together.[8]

If adequate reduction of LDL is not achieved with lovastatin, we use a combination of either nicotinic acid or lovastatin with one of the bile acid sequestrants. With combination therapy, lower doses of both drugs can be used, avoiding some of the dose-related

side effects. There are some very exciting studies using combination therapy that show actual regression of coronary artery disease.[9]

Summary

Cholesterol may be a more important risk factor for coronary heart disease in the elderly than previously realized. Lipid-lowering therapies, including diet and drugs, seem to be as effective in the elderly as in the young. However, it may take several years to see a significant decrease in cardiac morbidity and mortality. Therefore, therapy should be reserved for those with a life expectancy of at least ten years. In our experience, diet therapy is difficult for the elderly to follow. Drug therapy is well tolerated, if, as with any other drug, you start low and go slow.

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Promoting Successful Aging in the Primary Care Patient

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For the vast majority, aging is associated with the gradual decline of virtually all physiologic parameters (e.g., nerve conduction velocity), especially the ability of the individual to adapt to change, be it either internal (i.e., disease) or external (i.e., death of a spouse). Successful aging, on the other hand, represents the gradual adaptation to the effects of the passage of time with the relative maintenance of health.[1] The successfully aged individual remains fully independent and is relatively free from disease or disability. In this review, we will focus first on the risk factors for age-associated disease which should be altered (primary prevention), and then on the specific preventive measures (secondary prevention) of proven benefit.

The four major causes of death in those over 65 in this country are heart disease (51%), cancer (18%), cerebrovascular disease (16%), and influenza/pneumonia (4%), while the most common disorders affecting this age group are arthritis (44%), hypertension (30%), hearing loss (28%), heart disease (27%) and diabetes (12%). Clearly, tobacco use, improper diet, and lack of exercise are major risk factors for our elderly population.

The evidence is overwhelming that cigarette smoking is the single best external discriminator between health and disease.[2] Therefore, helping a patient stop smoking or chewing tobacco may have the greatest impact on future health.

Like tobacco use, the American diet is not conducive to healthy aging because it is excessively high in cholesterol, saturated fats and concentrated carbohydrates, and low in fiber. Both length of life[3] and health[4] are directly related to total caloric intake and dietary content. In view of the clear association of high cholesterol with heart disease, and hyperglycemia with neuropathy, our diet should consist of adequate but not excessive quantities of fruits, vegetables, fish, poultry and cereals. Total caloric restriction may give

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rise to a 7-10 year increase in maximum life span, and cholesterol restriction may add 5-10 years to average length of life.

Declining ability to run, climb stairs, or even walk is characteristic of age-associated disability. Our sedentary lifestyle contributes to obesity, atrophy of muscle mass and decreased joint flexibility. However, loss of muscle mass and joint flexibility is reversible. On an even higher level, declining functional aerobic capacity is also somewhat reversible.[5-7] The reality of physical functioning and aging is, "What you don't use, you lose."

To help patients eliminate the nicotine habit, change their diet or begin an exercise program, clinicians need to be committed to the goal of healthy aging in the hope of persuading their patients to commit to the same goal. Clinicians can assist the patient by providing education, setting a date to make a lifestyle change (e.g., quit smoking), assessing compliance and providing a referral to a support group if necessary.[8,9]

Another form of primary prevention is immunization. Influenza and pneumonia remain the fourth most common cause of death in the elderly, and, despite CDC recommendations for preventive therapy, vaccination is performed in the minority of elderly individuals. The protective effectiveness of Fluvax in the aged is 75% for community dwellers and 33% for nursing home residents.[10,11] The protective effectiveness of Pneumovax is 70% for community dwellers, 77% for low-risk nursing home residents, but may be as low as 0% for high-risk nursing home residents.[12,13] Clearly, therefore, virtually all elders should be immunized at least once with pneumonia vaccine and annually with influenza vaccine.

From the standpoint of specific screening tests that the busy clinician should provide to the asymptomatic elder, only those with proven effectiveness should be recommended. In this vein, an annual history, examination, electrocardiogram, blood count and metabolic screen is not warranted. However, there are interventions which effectively identify disease in its early stages.

In general, a screening test for preventive gerontology must be easy to administer, sensitive, and capable of preventing morbidity or mortality through early treatment of abnormalities. Clearly, exercise tolerance testing does not fit into this definition, and could not be recommended for routine screening of the aged. On the other hand, visual acuity testing is easy to perform and may identify the patient whose cataract extraction could prevent a fall or automobile accident. Therefore, simple assessments of vision, hearing and dentition or more detailed assessments such as mammography or Pap smears can and should be performed[14] (Table 1).

Table 1. Recommended Screening Tests for Asymptomatic Elders.[15]

Procedure	Men	Women	Interval
Visual acuity	*	*	annually
Auditory acuity	*	*	annually
Dental exam	*	*	annually
Breast exam/mammogram		*	annually
Abdominal exam for aneurysm	*		annually
BP measurement	*	*	every 2 years
Pelvic exam/pap smear		*	every 3 years
Weight	*	*	every 4 years
Cholesterol	*	*	every 5 years
Cardiac auscultation	*	*	once

In order to promote successful aging, we must first think about it. Furthermore, physicians themselves must set an example by living a lifestyle that promotes successful aging. Third, we must constantly encourage our patients to alter their lifestyle so as to decrease as much as possible their risk for dysfunction, disease or death.

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New Hope for Failing Hearts

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Major changes in our understanding of congestive heart failure have resulted from an etiologic shift, identification of neurohormonal effects, and a realization of the importance of diastolic dysfunction. Accordingly, management has changed. Digitalis should be avoided with diastolic dysfunction. When the dysfunction is systolic, a regimen of digitalis, diuretic and ACE inhibitor may be optimal.

TREMENDOUS change in our understanding of congestive heart failure (CHF) has occurred in the past decade. Coronary artery disease and idiopathic cardiomyopathy have replaced hypertension and valvular heart disease as the most common etiologies underlying this syndrome. Our understanding of CHF pathophysiology has also changed. We've become increasingly aware of the importance of diastolic dysfunction in patients presenting with classic CHF symptoms. Additionally, we've identified the deleterious effects of neurohormonal activation. This has sparked considerable research into neurohormonal antagonists, some of which have improved patient survival. Consequently, there is emerging a new prognostic hope for patients with this devastating condition.[1] This review focuses on the management of CHF, primarily on left ventricular failure (LVF), since it is the left ventricle that most commonly fails.

Differentiating Systolic/Diastolic Dysfunction

Left ventricular failure may be the result of either systolic or diastolic dysfunction.[2] Diastolic dysfunction is present in a significant proportion of patients who present with CHF symptoms. Typically, these patients are elderly women with underlying hypertension and/or ischemic heart disease. They have difficulties with diastolic filling of the left ventricle, resulting in elevated filling pressures with normal ejection fractions. These patients should not be treated with digitalis preparations. Reduced contractility is not the problem. Digitalis only increases the risk of toxicity without any benefit. Further, diuretic therapy must carefully balance symptom relief against the significant decreases in cardiac output that occur with vigorous treatment.

Systolic dysfunction is the result of decreased myocardial contractility resulting in a decreased cardiac output and an increase in intracardiac volume. The increased volume leads to increased left ventricular filling pressures and ultimately elevated pulmonary venous pressures causing the "backward" symptoms of CHF: dyspnea and orthopnea. The decrease in cardiac output causes the symptoms of "forward failure": fatigue and decreased exercise tolerance. Additionally, the reduction in cardiac output leads to an increase in sympathetic activity, activation of the reninangiotensin system (RAS) and an increase in arginine vasopression. The net result of neurohormonal activation, when sustained, is a deleterious workload on the heart.

Management: Two Main Goals

Management of the patient with CHF begins with a careful history, physical examination and selected use of the laboratory to identify reversible etiologies and exacerbating factors. Reversible etiologies and exacerbating factors are not uncommon in patients presenting with CHF. Consequently, it is important to exclude severe anemia (Hb $<$ 8 gms/dl), apathetic

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hyperthyroidism, Paget's disease, hypertension, thiamine deficiency and valvular abnormalities, among others. Identifying and correcting these abnormalities may significantly improve both symptoms and survival.

In particular, one must consider the possibility of coronary artery disease (CAD) in patients with mild CHF. Some univariate and multivariate analyses of prognosis have suggested that underlying CAD is associated with worsened survival.[3,4] Further, data from the Coronary Artery Surgery Study (CASS) suggests that patients with mild LVF and triple vessel disease may do better with surgical treatment than with medical management. Given that CAD is now one of the most common etiologies underlying CHF, patients with new-onset CHF should be evaluated for this etiology. An echocardiogram should be considered not only to exclude reversible etiologies and exacerbating conditions but to separate those with systolic dysfunction from those with diastolic dysfunction. Serum sodium, a potential marker for RAS activation, is prognostically and therapeutically useful. Lee and Packer found that patients with a serum sodium less than or equal to 137 mEq/L had a higher mortality rate than those with higher sodiums at 2 and 3 years followup.[5] Therapeutically, hyponatremic patients are more likely to develop hypotension and/or renal dysfunction when treated with angiotensin converting enzyme (ACE) inhibitors.

The two main goals in managing patients with congestive heart failure are the relief of symptoms and the prolongation of life. However, the achievement of these two goals does not occur in parallel. The prolongation of life which occurs with direct-acting vasodilators, such as the isorbide-hydralazine combination, has minimal impact on symptoms and no improvement on exercise tolerance. The relief of symptoms that occurs with diuretic therapy may, by neurohormonal activation, actually reduce long-term survival. Consequently, our approach to treatment must focus separately on both goals.

Current intervention is directed at abnormal hemodynamics. Therapy is aimed at improving pump performance, reducing workload, and reducing preload by controlling excess salt and water retention. Improving pump performance in the outpatient setting is best accomplished with digitalis glycoside.

Reduction of workload involves both nonpharmacologic and pharmacologic modalities. Depending on the symptomatic status of the patient, varying degrees of physical rest are required. Those with Class II CHF need only avoid strenuous activity, while those with Class III and IV require set rest periods to decrease cardiac work. The improved survival observed with several vasodilator regimens makes these agents an important therapeutic consideration.

Reduction of preload is accomplished by control of dietary salt and the judicious use of diuretic agents. Those with Class II CHF need only to avoid the addition of salt at the table, while those with class IV require careful regulation. Diuretics are aimed at keeping the patient free of symptoms and peripheral edema. Once achieved, the patient's weight is noted and becomes the target "dry" weight for future control. Diuresis beyond dry weight may lead to deleterious neurohormonal activation.

In the past decade there has been significant discussion over which pharmacologic agents should be used as first line therapy. Packer has recently proposed several criteria to help in identifying first-line agents.[6] He suggested that first-line therapy should provide rapid relief of symptoms, prolong survival, alter the natural history of CHF, and be safe and well tolerated.

The only class of agents capable of rapid symptom relief are diuretics. These agents have an effect within hours. Digitalis and the various vasodilator regimens offer symptom relief in days to weeks. Consequently, diuretics would seem to be an important component of first-line therapy. However, it is clear that diuretic therapy alone is unable to maintain patients in a symptom-free state. Digitalis or ACE inhibitors are also required. Recent meta-analyses have established the symptomatic benefit of digitalis in patients with CHF and normal sinus rhythm.[7]

With respect to survival, neither diuretics or digitalis alone have been shown to make a positive impact on survival. In fact, there is some concern that diuretics may reduce long-term survival by a number of mechanisms, including neurohormonal activation. However, all vasodilator trials demonstrating an improved survival have been conducted on patients already receiving diuretics and digitalis.[3,8] In comparing vasodilators, isosorbide-hydralazine was shown to improve survival but without significant improvement in exercise tolerance or functional status. The ACE inhibitors, in contrast, not only improved survival but improved symptoms and exercise tolerance and reduced arrhythmias. Consequently, ACE inhibitors may be preferred in patients with CHF and seem to work in 70-80% of those treated.

The only agents demonstrated to alter the natural history of CHF are the ACE inhibitors. In post-myocardial infarction patients treated with either captopril or a diuretic, those receiving the captopril had attenuation of left ventricular enlargement, while those on diuretics alone had progressive enlargement.[9]

With respect to safety and tolerability, diuretics are generally well tolerated as long as they are monitored closely for metabolic complications and titrated to maintain dry weight. Digitalis had adverse effects in 4% in a recent multicenter trial.[10] If followed carefully, the risk of serious toxicity is low. Of the vasodilator regimens, isorbide-hydralazine was not tolerated in almost 38%. This is less of a problem with the ACE inhibitors, especially when small doses are used initially. Those with hyponatremia, those with ortho-

static hypotension, and those on large doses of diuretics require close monitoring of therapy with ACE inhibitors.

From the above discussion it is apparent that no one class of agent fulfills all the criteria of a first-line agent. Multiple agents are usually required. The combination of diuretic and ACE inhibitor is equally effective in controlling symptoms when compared to a diuretic and digitalis. However, prolongation of survival has only been demonstrated with all three agents. Therefore, therapy with all three agents with the smallest doses necessary to control symptoms is a reasonable approach for most patients with systolic dysfunction of the left ventricle.

Summary

This review has examined current approaches to therapy of CHF and emphasized the importance of separating those with systolic dysfunction from those with predominately diastolic dysfunction. Further, the importance of neurohormonal activation to patient decompensation was identified, as was the new hope generated by antagonizing this activation.

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Primary Care and Cancer in the Elderly

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MALIGNANT disease accounts for about one-quarter of all deaths in the elderly. Most cases of cancer are diagnosed by a primary care physician, and many, such as skin cancers, are readily cured and are not reflected in mortality rate statistics. After the initial diagnosis, many patients will be referred to medical or surgical oncologists, but in most cases the primary care physician will continue an important role in the care of the patient. Certainly the patient and family hope that the continuing care relationship with the physician they know best will continue through what may be difficult times.

This paper concerns itself with selected aspects of the role of the primary care physician in cancer prevention and diagnosis, beginning with some considerations of the epidemiology of the cancers most common in the elderly, especially those which continue to increase in incidence with advancing age. The present status of early diagnosis of the major cancers will be considered, and some possibilities of prevention will be included.

Epidemiology

Of most importance are those malignancies which 1) occur more often in older rather than younger persons and 2) which occur with continuously increasing incidence with advancing age. Although we generally consider that cancer is a problem of advancing age, there are tumors which are more common in young or middle-aged persons. Examples include testicular tumors, which are more common in young men; acute lymphoblastic leukemia, which is more common in children; carcinoma of the uterine cervix, which peaks in younger women; or brain malignancies, which are less commonly seen in patients in the older age group.

The incidence of many common tumors of the elderly is shown in Table 1. These data, for males and females over the age of 65, are derived from the Surveillance, Epidemiology, and End Results (SEER) surveys of the the National Cancer Institute.[1] By far the most common malignancy in men is prostate

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Supported by Grant 5K07AG00404 of the National Institute on Aging.

Table 1. Age Adjusted Annual Rates/100,000 Whites Over 65.

Primary Site	Male	Female
Prostate	635	—
Lung and bronchus	482	129
Breast	4	332
Colon and rectum	404	289
Urinary bladder	186	45
Uterine corpus	—	102
Stomach	81	35
Mouth, tongue, pharynx	74	26
Pancreas	69	49
Leukemia	69	34
Hodgkin's disease	66	41
Lymphoma	61	48
Kidney, urinary tract	61	25
Larynx	41	6
Liver, biliary tract	35	28
Myeloma	26	19

cancer. Its clinical impact does not quite reflect this since a number of small tumors are found incidentally at prostatectomy or at autopsy. Cancer of the lung and bronchus, the leading cause of cancer deaths in this country, still has a greater incidence in men over women, reflecting the fact that smoking in men became commonplace a generation before it did in women. Although breast cancer still leads in *incidence* in women, very shortly lung cancer will overtake breast cancer as the leading cause of cancer *deaths* in women. If the prevalence of smoking continues to fall dramatically among men while remaining stable in women, as the trends suggest, in the future lung cancer deaths will be more common in women than men.

Hodgkin's disease is a tumor which seems to have two peaks in its curve of incidence. The first peak occurs in persons in their 20s and 30s, and there is another increase in incidence in those over the age of 60. The older patients are more likely to have the more malignant (and less curable) lymphocytic depletion form.

Looking at the incidence of lung cancer with age, it seems to decrease after the age of 60. However, if only smokers are included in the data, the incidence rises continuously with age.[2] Carcinoma of the prostate continues to increase in incidence with age, and in the 85+ age group, the incidence is over 1000/100,000 per year.[3] Interestingly, leukemias, generally thought of as rare disease, increase exponentially after the age of 50, so that at age 85, the rate is 150/100,000/year in males.[3] This group then becomes as common as pancreatic or laryngeal cancer. In fact, if all the hematopoietic neoplasms are added together, their incidence makes them, rather than rare problems, important causes of morbidity as the population ages.

Screening

Various organizations, such as the National Cancer Institute and the American Cancer Society, have publicized guidelines for screening for cancer. The guidelines sometimes vary between groups, especially in different countries. All represent attempts to propose cost-effective measures which save lives and do not siphon off scarce health-care dollars needed for many other purposes.

The broad aim of screening is to lower the death rates from cancer. Useful screening has two main requirements: 1) it must be shown that the tumor can be more effectively treated if found early, and 2) the screening measure(s) should be accurate and cost-effective. Although we intuitively feel that certain screening measures, such as digital rectal exams and testing for occult blood in the stool, are useful, recent study panels have looked for actual hard evidence for efficacy of many screening procedures.

The National Cancer Institute has studied these issues and found that available evidence shows efficacy for some tumor screens but not others.[4] There is hard evidence for only two tumors that screening can reduce cancer mortality: 1) breast cancer screening in women over 50, and 2) screening for cervical cancer. For lung cancer, evidence shows that screening does not reduce mortality, and the evidence is inconclusive that screening reduces mortality for a number of common tumors. These include colorectal cancer, bladder cancer, breast cancer in women aged 40-49, prostate cancer, oral cancer, melanoma, endometrial cancer, esophageal, and gastric cancer.[4]

A recent comprehensive publication on screening and prevention of 60 common diseases has been published by the U.S. Preventive Services Task Force.[5] Their recommendations concerning tumors common in the elderly are included in Table 2.

These recommendations are generally less demanding than previous recommendations by the National Cancer Institute and the American Cancer Society. This leaves the practitioner in something of a quan-

Table 2. Prevention of Cancer 1991.

Breast Cancer: 1) annual clinical breast examination; 2) mammography every 1-2 yrs between 50-75 yrs of age; 3) breast self-examination

Colorectal Cancer: 1) insufficient evidence either for or against fecal blood testing or sigmoidoscopy in asymptomatic patients but no basis to discontinue their usage

Lung Cancer: routine chest x-ray or sputum cytology not recommended

Prostate Cancer: 1) insufficient evidence for or against rectal exam as effective; 2) ultrasound and serum markers not routinely recommended

Cervical Cancer: 1) Pap smears every 1-2 yrs but discontinue at age 65 if consistently normal

Skin Cancer: 1) routine screening recommended for persons at high risk; 2) advise sunscreens; 3) no evidence for or against self-examination

Oral Cancer: 1) routine screening not recommended; 2) carefully examine tobacco and alcohol abusers; 3) counsel patients to have regular dental examinations, to discontinue tobacco use and limit alcohol intake, and to apply sunscreen on lips and skin for sun exposure

Pancreatic Cancer: routine screening not recommended

dary. For instance, even though there are no firm conclusions to be drawn from existing data concerning efficacy of digital rectal exams as a cost-effective measure, most practitioners will continue to include this as a regular part of complete examinations. Likewise, although routine screening for oral cancer is "not recommended," it is certainly simple, inexpensive and reasonable to examine the oral cavity carefully in older persons. Each physician should be aware of the guidelines, and make appropriate clinical judgments about their utilization.

The Cancer Control Program of the National Cancer Institute, originally initiated in 1971, has recently proposed dramatic new cancer control objectives for the year 2000.[4] They envision a 50% reduction in cancer mortality rate by the year 2000 compared to rates in 1990. This seems remarkable in view of the somewhat disappointing finding that many early screening efforts may not be truly effective, and the continuing difficulties in curing or treating some of the most common malignancies, such as lung and colon cancer.

The expert panels of the NCI feel, however, that the goal can be reached with two important developments. First, since lung cancer is the most important cause of cancer death, a significant reduction in that one tumor would greatly improve overall cancer mortality rates. Thus a goal of reduction of the percentage of adults who smoke from 34% to 15% or less, and a reduction in the percentage of youths who smoke by age 20 from 36% to 15% or less are included. The other major goal (or assumption) is that the historical incremental decrease in mortality rates for all major cancers will continue to improve by 1.5% each year until the year 2000. A hopeful sign in this is that between 1982 and 1984 (the latest data), there has been a yearly small decline in incidence rate for lung cancer in white males.

Whether such remarkable changes will truly occur in cancer incidence in that short time span will depend on dramatically effective public education programs but will also require continuing efforts by the patients' primary care physicians in counseling them about preventable disease.

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Ethical Dilemmas in Geriatric Care

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ALTHOUGH written in the 5th century BC, the principles raised in the Hippocratic Oath still govern our current ethical conduct. Hippocrates urged his students to share acquired knowledge, to use acceptable measures to benefit the sick and to keep them from harm and injustice. The Oath places heavy emphasis on professional conduct; it should at all times embody a sense of justice, obligation and personal decorum. Predating societal recognition of individual rights by over a thousand years, Hippocrates regarded all details of illness and treatment as confidential between patient and physician.[1] The new field of biomedical ethics, emerging in the last 25 years, includes issues related to biomedical research and the societal problems of medical technologies. Some ethicists have further narrowed the field to include "clinical ethics", the identification, analysis and resolution of ethical problems in the care of patients in the context of "shared" decision making.[2] These decisions are based on four ethical principles:[3] 1) *nonmaleficence*, the duty or obligation not to cause harm to patients; 2) *beneficence*, the duty or obligation to benefit patients and to help them further their own legitimate interests; 3) *autonomy*, the duty or obligation to respect persons and promote self-determination by patients; 4) *justice*, the duty or obligation to allocate social burdens and benefits fairly among all persons.

Biomedical ethics and geriatrics have shared a parallel exponential growth over the past 15 to 20 years. This development is a result of increased utilization of medical technology and better living conditions. The average life span has increased, thereby leading to an elevation in the absolute number and proportion of persons aged 65 and older. Whether recognized or not, health care rationing is a reality, and, although a formal system is still evolving, it is mandatory that physicians responsibly utilize health care dollars. We know that most health care dollars are spent on the last 24 hours of life. We must ask ourselves, Are we

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guiding our patients down an expensive and futile path because, as one physician writes, "death was a defeat, a failure; a chronic disease, a constant reminder of the physician's impotence."[4]

The following cases illustrate important issues.

Case Report One

Mr. V.B. is a 74-year-old male with Stage D prostate cancer. He has spinal cord compression with secondary paraplegia and incontinence. His hospital course has been marked by frequent bouts of urosepsis and pneumonia. His wishes regarding CPR were discussed with him, and he requested to be resuscitated at all costs and maintained on technology without consideration as to his quality of life.

This case demonstrates that patient autonomy may conflict with what some physicians perceive to be futile therapy. The goal of any treatment should be to improve the patient's prognosis, comfort, well-being, or general state of health. This goal is clearly in conflict with futile treatments. Patients or surrogates should be informed of the futility judgement even though consent of the patient is not required. Others have recommended that care givers have no obligation to discuss useless therapy.[5] Futility describes any effort to achieve a result that is possible, but that reasoning or experience suggests is highly improbable, and that cannot be systematically produced. Futility is a professional judgement that takes precedence over patient autonomy and permits physicians to withhold or withdraw care deemed to be inappropriate without subjecting such a decision to patient approval.[6]

Futility relates to the elderly most poignantly in the area of resuscitation. Sudden cardiac death accounts for 400,000 to 600,000 deaths each year in the United States. Most cases occur out of hospital and few live to be admitted. For out-of-hospital cardiac arrest, survival to discharge is less than 1%. For in-hospital arrest, survival to discharge is only 6.5%. Interestingly, in a study of 503 charts, 96% of patients who had a cardiac arrest did not document that a CPR discussion had taken place prior to the event, 2.8% of the patients had discussed resuscitation with their physicians and wanted it, four patients requested CPR with no intubation, and three were resuscitated despite a previous DNR order.[7,8]

Although difficult when the patient and physician have not had adequate time to establish a relationship of trust, a straightforward discussion about the patient's prognosis, the expectations and realities of available care, and recommendations as to the appropriateness and likelihood of success of various treatments is critical.[9]

Case Report Two

Mr. T.R. is a 65-year-old male with a history of metastatic squamous cell carcinoma of the lung. He developed post-obstructive pneumonia, was intubated and later found to be ventilator dependent. The patient was competent and requested that life support be discontinued and he be allowed to die. The family was in agreement, and the patient expired shortly after the ventilator was turned off.

The second case demonstrates the rights of the individual to refuse life-sustaining treatment after being made aware of his/her prognosis. This type of informed consent is a constitutional right. As stated by the courts in *Cruzan vs Director, Missouri Department of Health* ". . . for purposes of this case, we assume that the United States constitution would grant a competent person a constitutionally protected right to refuse lifesaving hydration and nutrition."[9,10] Recognition of the concept of autonomy has led to development of advance directives. These are medically and legally binding documents that allow patients to express their wishes regarding medical care.

To aid your patients in making an informed decision, it is recommended that you document in the medical record the patient's feelings about various modes of treatment. Living will forms should be available, and surrogate decision-making should be explained.

In summary, there truly is an art to shared decision making. A mutual understanding between patient autonomy and beneficence on the part of the physician must occur. Proper care of patients at the end of life will require that we overcome the denial of death and our addiction to technology. Hard-won experience forces physicians to learn more about and incorporate into patient care the aspects of healing beyond the reach of technology.

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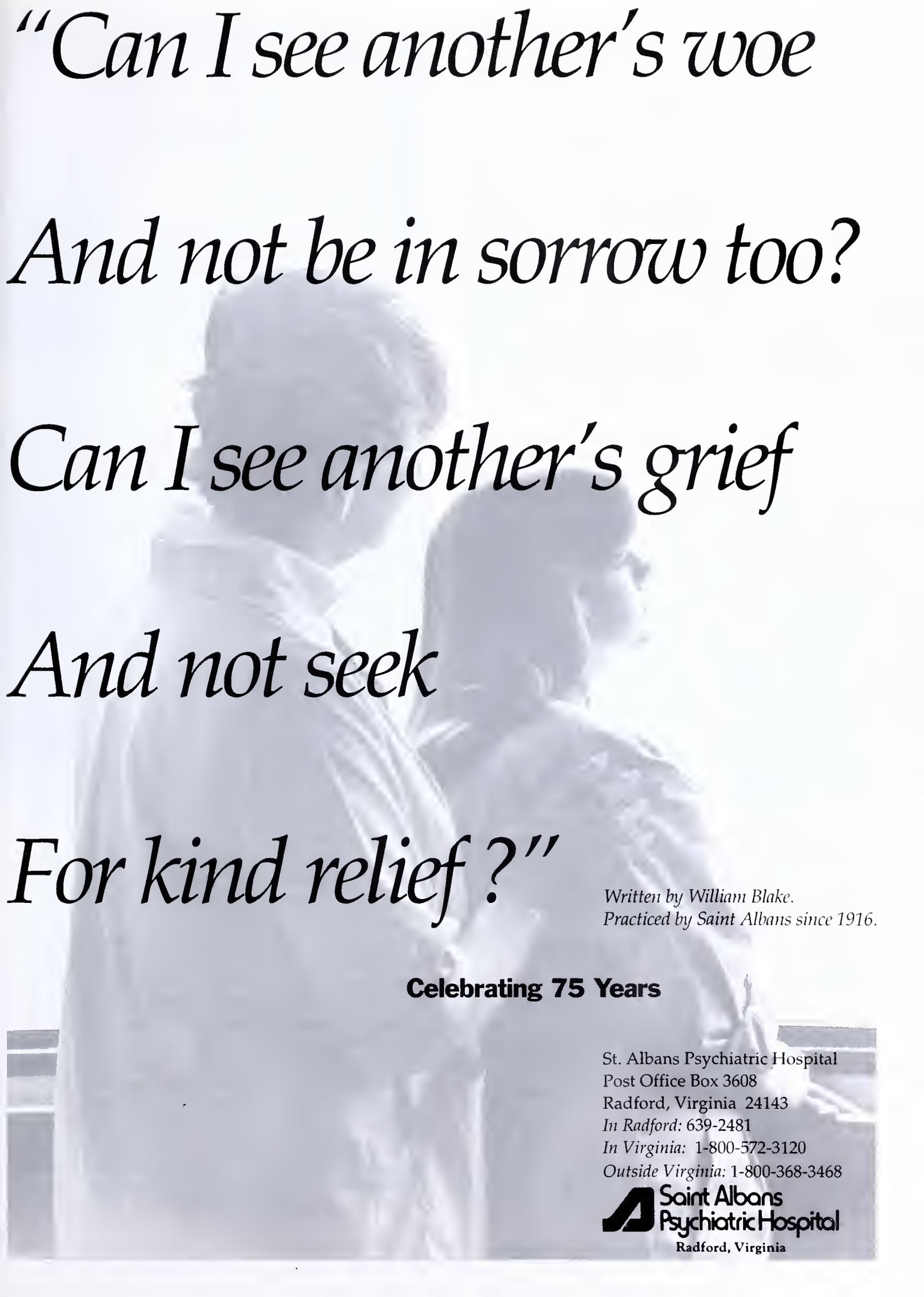
"Can I see another's woe
And not be in sorrow too?
Can I see another's grief
And not seek
For kind relief?"
Written by William Blake.
Practiced by Saint Albans since 1916.
Celebrating 75 Years
St. Albans Psychiatric Hospital
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Radford, Virginia 24143
In Radford: 639-2481
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Saint Albans
Psychiatric Hospital
Radford, Virginia

Cosmetic Surgery of the Aging Mouth

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A FACE LIFT can get rid of a drooping neck and heavy jowls, making a person look younger, and trimming away the sagging skin and baggy fat of the eyelids can make a person look wider awake and healthier, but the mouth area, untouched, will still look old.

There are many changes that occur around the mouth with aging. The lip lengthens, the vermilion thins out and takes on a "set" look, the corners turn down, wrinkles occur. The upper teeth stop showing at all when the face is in repose and show less with smiling. In the last decade a number of procedures have been developed to correct aging of the mouth. This paper presents two particularly effective ones. Both are simple skin excisions and repairs.

The Lip Lift

By shortening the central part of the upper lip, the teeth show when the lips are lightly separated (Figs. 1A and 1B).

Technique. A wavy ellipse is marked out under the nose following the contour of the nose-lip junction. Averaging 6mm wide, it may be as narrow as 3mm or as wide as 11mm, depending on an artistic sense of how much to shorten the upper lip to mimic the shorter lip of youth, one that shows 3-4mm of tooth margin in repose. A good rule of thumb is to over-correct by about one-third to allow for some postoperative re-droop. This strip of skin is excised, and after cautery of the bleeders, precise repair is made with interrupted subcuticular sutures of 6-0 Vicryl. External skin sutures are seldom used. A more technical description of the procedure has appeared elsewhere.[1]

Results. Between April 1980 and April 1990 my associate and I performed 285 lip lifts. Early on, 3-4mm were excised, but it wasn't enough and several patients requested more lifting. Now I remove 6-8mm or more and find it gives a better appearance.

It is difficult to provide measurements for comparison because the results lie in the subjective realm of aesthetics and the patient's personal satisfaction, and because there is little in the literature with which to compare. Additionally, the procedure has seldom been performed alone but has usually been adjunctive to a face lift.

It can be reported, however, that of the 285 patients, 281 were satisfied with the procedure. Two of these looked uneven afterwards, the lip lift worsening a preexisting asymmetrical height of the lip, but both of these were corrected.

Of the remaining four, two felt "too much" had been taken and two felt they looked worse. One of the "too much" patients wrote at six months to say she had adjusted to it and was pleased. The other was not heard from again. Both of the "made-me-look-worse" patients were early in the series and revealed a particular problem—the mouth with downturned corners. Elevating the central part of the lip with the lip lift emphasized these downturned corners, so the corners had to be elevated. Thus was created the "corner lift," which worked so well that I began to notice how many older people have this problem.

The Corner Lift

By elevating the corners of the mouth the look is shifted from angry or unpleasant to serene (Figs. 2A and 2B).

Technique. Mark a dot at the commissure. Mark out a triangle of skin just above each commissure by 1) extending a line medially from the dot along the skin-vermilion junction for about 12-16mm; 2) extend a line from the dot aiming at the top of the ear, stopping short of ther nasolabial crease; and 3) connect the ends of the two lines to form a triangle. (The height of the triangle will depend on the amount of elevation—3mm for a minimal amount, which probably is not worth the scar tradeoff, 5mm for the usual amount, and 7-9mm to correct a more significant downturn.) Excise the triangle of skin. Suture precisely in two layers. I use subcuticular sutures of 6-0 Vicryl and 6-0 nylon skin sutures. Remove the skin sutures in 3-5 days. The short scar loses its pinkness and firmness over three months and covers fairly easily with makeup during this time. The final white line is hard to see and lies at or slightly within the lipstick border.

Results. During the five and a half years January 1985 to July 1990, we performed corner lifts on 277 patients. The downturned look disappeared in all patients, and for some, depending on how much was excised, there was even a slight upturn, prompting one patient to refer to the procedure as the "Mona Lisa correction."

Only one patient of the 277 developed a poor scar, on both sides of an oily-skinned male. It disturbed the surgeon (but not the patient), but a revision did not narrow it. Eight patients had slightly depressed scars and requested they be filled in. This was done with collagen or a microdrop of medical-grade liquid silicone. Only one patient disliked the result, a male whose *inner* anger was unchanged.

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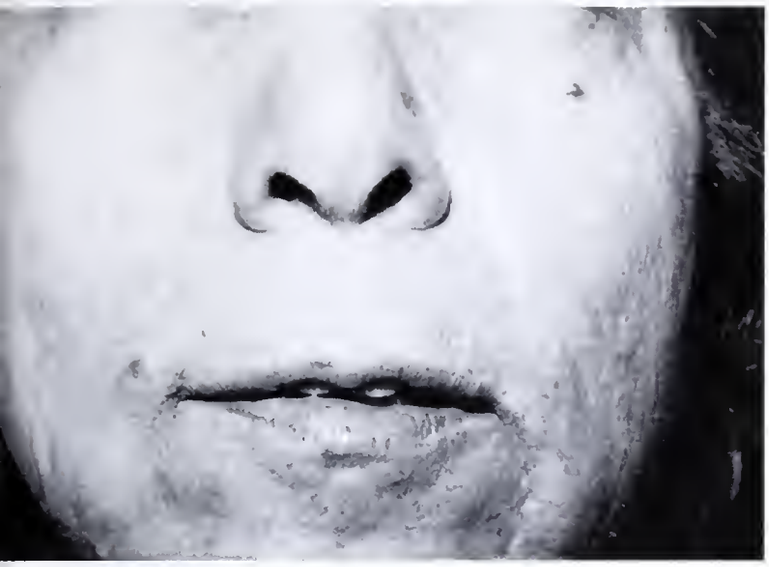
Submitted 8-12-90.



Figs. 1A and 1B. Lip lift in 50-year-old patient. Left, preoperative view; right, five weeks postop.

Figs. 2A and 2B. Corner lift in 50-year-old patient. Left, preoperative; right, 2½ months postop.

Discussion

The legitimacy of cosmetic surgery to correct aging is based on certain premises. Among them are 1) that our culture has a bias against aging, and although it is stronger against women, it is damaging to men also; 2) that regardless of chronological age, there is an inner age seldom exceeding 35-40, and it is all right to try to look as young as one feels. The results of rejuvenation surgery in my experience are generally excellent and they are becoming more consistent as the specialty matures.

Although objective results are obvious, the subjective results are equally if not more important. Patients almost uniformly report that "I feel better about myself." The office staff observes that these patients walk with a springy step, wear brighter colors, their speech is more animated.

The mouth ages markedly. Almost one-half of the patients who show up as possible candidates for rejuvenative surgery have some concern about the appearance of the mouth, yet less than 5% of the pages of the two most widely-read texts on cosmetic surgery[2,3] are devoted to the mouth area. The aging mouth needs more attention paid to it.

There are, of course, other techniques of perioral rejuvenation, including dermabrasion for wrinkles, various techniques for making the lips fuller, and chin implants to help correct the drooping "witch's chin." The two techniques presented here have proven simple and effective and have had excellent patient acceptance.

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ABSTRACTS

These are abstracts of papers to be presented at the annual meeting of the Virginia Surgical Society to be held May 3-5 in Williamsburg. Dr. Irving L. Kron is program chairman.

Review of Laparoscopic Cholecystectomy Performed in Community-Based Hospitals. Randolph J. Gould, MD, and Lynne P. Clark, MD, Norfolk.

The experience of laparoscopic cholecystectomies performed by multiple independent surgeons in community-based medical-school-affiliated hospitals is reviewed. This study examines the indications for laparoscopic cholecystectomy and the acceptable morbidity. Intraoperative cholangiography, pre- and post-operative ERCP, use of lasers, and surgical credentialing will be discussed.

Two hundred operations were performed during the period of March-October 1990. The ages of the patients were 18-81; 140 females, 60 males. Symptoms were present from one week through ten years. 84 patients (42%) had undergone previous abdominal surgeries. 141 operations (70%) were performed in less than two hours. Twenty patients (10%) underwent intraoperative cholangiograms. Nine patients (5%) were converted to open procedures: 7 for porcelain gallbladder, empyema, acute inflammation with difficult dissection and impacted stones in cystic duct; 2 were opened for common bile duct injury and enterotomy. The CBD injury was treated with choledochojejunostomy with Roux En Y loop. The enterotomy was treated with resection and primary repair. Both of the postoperative courses were unremarkable. Nine patients (5%) underwent preoperative FRCP for hyperamylasemia, elevated LFTs or dilated CBD on ultrasound. Only one patient revealed CBD stones, which were easily retrieved.

Postoperative complications included one bile leak requiring ERCP with documentation of cystic duct remnant leak treated with nasobiliary stent without sequelae. Six patients (3%) reappeared with abdominal pain and/or jaundice. These patients underwent ERCP. Five patients had retained stones which were retrieved endoscopically. 128 patients (64%) were discharged within 23 hours of admission.

This study to date indicates that laparoscopic cholecystectomy performed by appropriately trained surgeons is a safe procedure with an acceptable morbidity rate. Data continues to be collected on a larger patient population.

Laparoscopic Cholecystectomy: Safe and Effective Treatment for Symptomatic Cholelithiasis. Bruce D. Schirmer, MD, Janet Dix, PAC, Stephen B. Edge, MD, Matthew J. Hyser, MD, John B. Hanks, MD, and Manuel Aguilar, MD, Charlottesville.

From February 1990 to January 1991, 190 patients referred to the Gallstone Treatment Center at the University of Virginia have undergone treatment for symptomatic cholelithiasis using laparoscopic cholecystectomy (LC). During this period the number of surgeons performing LC has grown from 2 to 5. The patient population included 44 males (23%) and 146 females (77%). Average age was 44.1 ± 1.1 years (range 17 to 83). Average weight was 172.6 ± 3.4 pounds (range 75 to 365). Gallstones were diagnosed by preoperative ultrasound in 179 patients (94.2%). Most patients (66.3%) had multiple gallstones, but 21.6% had a single stone and 7.4% had 2 or 3 stones only. Average time of operation was 137 ± 3 minutes (range 52 to 320). Intraoperative cholangiography was attempted in 78% of cases and successfully completed in 66% of attempts. LC was successfully completed in 90.5% of all cases, while 9.5% of cases required conversion to open cholecystectomy. There has been no operative mortality. The morbidity rate was 3.7% for significant and 6.3% for minor complications. Patients undergoing successful LC stayed an average of 1.26 ± .10 days. One third of patients (33.2%) undergoing successful LC required no pain medication after leaving the recovery room. At the present time the only absolute contraindications to attempting LC in our experience are pregnancy and large common duct stones. We conclude LC is a safe and effective method of treating almost all patients with symptomatic cholelithiasis.

Stapled Ileoanal Anastomosis for Ulcerative Colitis and Familial Polyposis Without a Temporary Diverting Ileostomy. Harvey J. Sugerman, MD, Heber H. Newsome, Jr., MD, Alvin M. Zfass, MD, and Gayle DeCosta, RN, Richmond.

British studies have proposed a transabdominal stapled ileoanal procedure for ulcerative colitis (UC) and familial polyposis (FP). Since March 1989, we have performed 20 stapled ileoanal procedures (19 UD, 1 FP) without ileostomy in 18, of whom 13 were taking prednisone and 8 underwent semi-emergent surgery for uncontrollable bleeding. Fifteen pts have been followed > 4 months. During the same time period, an additional 4 pts required a standard ileoanal procedure. The results were compared to 25 pts who previously underwent mucosal stripping and sutured J-pouch ileoanal anastomoses with a temporary ileostomy.

Results: Non-Stapled (NS) Group: 3 pouches ex-

cised (1 complications, 2 excessive stool frequency), 1 pelvic abscess, 1 stenosis requiring dilatation under anesthesia, 1 enterocutaneous fistula after ileostomy closure, 1 ileostomy site hernia, and 2 small bowel obstructions. Stapled (S) Group: 1 anastomotic leak, 1 pouch leak, 1 pelvic abscess. Pts were managed successfully with drainage (all 3) and diverting ileostomy (1). One pt developed stenosis requiring dilatation under anesthesia. Stool frequency was similar in the two groups, NS = 8.0 ± 4.6, S = 7.5 ± 2.5.

	Resting Pressure	Accidents		Spotting		Protective Pad
		Day	Night	Day	Night	
S	46 ± 13 mmHg*	12%*	18%**	24%*	35%*	6%**
NS	34 ± 13 mmHg	41%	65%	63%	69%	47%

*p<0.05; **p<0.01

All but one UC patient had residual disease at the anastomosis. Anal mucosa between the dentate line and stapled anastomosis was 1.8 ± 1.3 cm (range 0 to 3.5 cm).

Conclusions: The stapled ileoanal anastomosis is a simpler, safter procedure with less tension but leaves a small cuff of residual disease. It provides significantly better stool control and may permit avoidance of an ileostomy with its complications.

Quadruple Loop (W) Ileal Pouch with Ileal Pouch/Anal Anastomosis: Improvement in Functional Results after Proctocolectomy for Ulcerative Colitis. David R. DeHaas, Jr., MD, Virginia Beach.

Following proctocolectomy, ileal pouches of the J-shape, S-shape, and W-shape have been described to preserve continence with the J-pouch gaining popularity in this country. Large series of J-pouch patients still report significant incontinence rates and stool frequency rates of 6 or more stools per 24 hours. This report analyzes 13 patients who underwent W-ileal pouch reconstruction after total proctocolectomy for ulcerative colitis. Patients ranged in age from 23 years to 62 years with 5 patients of 40 years or older. There were no deaths or significant perioperative morbidity. Following takedown of loop ileostomy, all patients were noted to be completely continent, both during the day and at night. Average stool frequency was 3.5 times in 24 hours. All patients have been able to evacuate their pouch spontaneously and only one patient has experienced pouchitis. Anal manometry was performed preoperatively and postoperatively on the past 9 patients. Loss of the normal relaxation reflex of the internal sphincter was a consistent finding in the postoperative study, but good preservation of external sphincter function was noted in all patients. These results suggest that the W-pouch reservoir, although more tedious to construct, may offer improvements in stool frequency rates and in continence over the J-pouch design.

Early Experience with Vascular Endoscopy. F. Noel Parent III, MD, Jock R. Wheeler, MD, Roger T. Gregory, MD, Stanley O. Snyder, Jr., MD, and Robert G. Gayle, MD, Norfolk.

Flexible fiberoptic technology has led to the development of several small diameter endoscopes for use in arteries and veins. The utility of vascular endoscopy in 15 patients is reviewed. Angioscopic survey of saphenous vein bypass grafts was performed in 8 patients. Incompletely cut valve leaflets were identified in 3 patients and was associated with a particular valvulotome in each instance. Intimal injury was identified in one patient. The distal anastomosis was assessed in selected patients with large (3mm) vein grafts. In one patient, the in situ bypass surgery was performed without the standard length thigh incision by employing a Hall valvulotome in a "blind" fashion and ligating venous tributaries through small skin incisions after angioscopic localization. Endovascular intervention such as atherectomy devices and balloon angioplasty were evaluated in seven patients. Two patients undergoing PTA alone were observed to have intimal dissections of plaque. Five patients treated with atherectomy devices (2 Kensey catheter, 3 IVT/TEC) were examined by angioscopy and found to have residual disease not appreciated by angiography. Thromboembolectomy was aided by angioscopic localization of thrombus in one patient. In another patient, angioscopy diagnosed white clot syndrome as a cause of graft failure, which was treated with urokinase infusion.

In our experience, angioscopy has proven to be a sensitive diagnostic and descriptive tool which has advantages over conventional methods of vascular assessment. Angioscope assisted in situ saphenous vein bypass surgery is an attractive alternative to standard techniques and deserves additional investigation.

Acute Gastrointestinal Complications Following Aortic Surgery. Bruce A. Mast, MD, Hunter H. McGuire, Jr., MD, and Michael Sobel, MD, Richmond.

Gastrointestinal complications following aortic reconstruction are reported to occur in 7% of patients, more commonly following surgery for aneurysmal disease. To better define the types of complications, their relationship to the vascular procedure, and to identify causative and preventable factors, all acute gastrointestinal complications (GIC) occurring after aortic surgery at the McGuire Veterans Administration Medical Center during a 13-year period (1977 to 1989) were studied. A summary of key data is provided below:

	N	Pts	GIC	VI	Mortality	
Aneurysm	289	5	5 (1.7%)*	4 (1.4%)	4/5 (80%)	(*p<0.025 by
Occlusive	275	11	14 (5.1%)*	6 (2.2%)	6/11 (55%)	chi square)

(N=total patients; Pts=patients with GIC; VI=visceral ischemia; Mortality=deaths of patients with GIC)

Continued over

There were significantly more GICs following surgery for occlusive disease, mainly attributable to nonvascular problems, including bowel obstructions, iatrogenic colonic perforations, an abscess following concomitant cholecystectomy, pancreatitis and gastric ulcer. Neither emergent surgery, preoperative hypotension nor massive transfusion were predispositions to GICs. Refractory postoperative hypotension, embolism from atrial fibrillation and surgical technical difficulties were identified as contributory.

In contrast to previous reports, the overall incidence of GICs was only 3.3%, although the complications were significant as indicated by their high mortality. The incidence of visceral ischemia was similar in both patient groups, but overall, GICs occurred more frequently following reconstruction for aortic occlusion. This may reflect the generally poorer state of health in these patients with systemic atherosclerotic vascular disease. Meticulous surgical technique, avoidance of ancillary GI surgery (e.g., cholecystectomy) and aggressive management of postoperative cardiovascular events may prevent many of these complications.

Late Complications of Blunt Gastrointestinal Trauma. Wayne H. Wilson, MD, Salem, and Worthington G. Schenk III, MD, Charlottesville.

Intestinal trauma has the mystique of being a rare event when dealing with blunt trauma but in reality is frequently present as a serious injury. Current management generally mandates definitive care within 10-24 hours as the accepted standard of treatment. Even when this goal is not achieved, a review of the literature shows that if significant injury has occurred, that it generally becomes manifest and is treated within two weeks of the initial trauma. This paper discusses a patient managed at the Salem Department of Veterans Affairs Medical Center who required surgery 34 days after his initial injury because of gastrointestinal obstructive symptoms. This case prompted a review of the English literature since 1930 and identified 32 patients in which long-delayed complications ensued and only then became recognized and were surgically corrected. The commonest indication mandating surgery was gastrointestinal obstructive symptoms. The most frequent lesion identified during laparotomy was cicatricial stenosis. Biomechanics and pathogenesis of the injury are discussed as well as the epidemiology, etiology and location of the injuries along the gastrointestinal tract. The small intestine was the most frequent site of this type of injury followed by the colon. The average lapse of time before surgical intervention occurred was 66 days.

Two points seem worth emphasizing: 1) in 80% of the cases the patients had ongoing symptoms from the time of injury; 2) in the cases in which pathology was identified and followed, the process did not abate, surgery was ultimately required, and in one instance, emergency surgery was precipitated while the lesion was being followed. We conclude that late complications of blunt gastrointestinal trauma, although infrequently reported, must be considered and when identified require surgical correction.

HIDA Scanning in Complications of Biliary Trauma. P. Scott Seibel, MD, Carol M. Gilbert, MD, and Robert E. Berry, MD, Roanoke.

Posttraumatic biliary leak may be an insidious problem which presents a diagnostic dilemma to the clinician. Radiopharmaceuticals offer a noninvasive diagnostic test for evaluating difficult clinical problems connected with hepatobiliary disorders. HIDA scintigraphy is used extensively in evaluating hepatic transplants, cholecystitis, and other nontraumatic diseases of the liver and biliary tract. We present two cases in which this technique was helpful in the detection of complications of liver injury by demonstrating not only abnormal fluid collections but also identified the site of the leak. While other diagnostic modalities, including CT scan, paracentesis, arteriography and ultrasound, are used in the evaluation of biliary trauma, each has its particular limitations. The HIDA scan is noninvasive, widely available and easy to perform, making it a first-line approach for those

patients suspected of having disruption of the liver or biliary tree.

Management of Sacral Chordoma. Kevin P. Bethke, MD, and James P. Neifeld, MD, Richmond.

Chordoma is a rare malignant tumor derived from the primitive notochord and is located along the axial skeleton. Traditionally it has been considered a slow-growing, relatively silent, locally aggressive tumor with little potential for distant metastasis. During the past 20 years we have treated 15 patients with chordomas: 8 originated in the sacrum, 3 in the clivus, and 4 elsewhere along the axial skeleton. This report will focus on those patients with sacral chordomas.

Pain was the most common presenting symptom. The insidious nature of the tumor is demonstrated by the long interval between the onset of symptoms and diagnosis (median of one year with a range of 4 months to 6 years); 2 patients had undergone coccygectomies and one patient a lumbar discectomy prior to establishing a diagnosis of sacral chordoma.

Seven patients underwent resection and one patient received primary radiation treatment only. Four of the resections were done initially at the Medical College of Virginia, while 3 patients were referred for resection of recurrent tumor. The median disease-free survival was 3.0 years (range 1.5 to 6.5 years) and the median overall survival was 4.2 years (range 1.5 to 11 years). Three patients had distant metastatic disease at the time of death.

These data demonstrate the need to include chordoma in the differential diagnosis of low back and sacral pain. Early diagnosis and aggressive surgical treatment are necessary to provide the best chance for long-term survival.

The following abstract derives from an exhibit presented at the annual meeting of the Medical Society of Virginia held November 1-3 at the Homestead.

Cytopathology and Histology in Clinically Single or Dominant Thyroid Nodules. Alton R. Sharpe, Jr., MD, and William J. Frable, MD, Richmond.

From 1980 to 1984, 195 clinically single or dominant thyroid nodules were examined by thin-needle aspiration (TNA) biopsy cytology. Forty-five of these patients were also examined histologically. Malignant thyroid nodules were histologically confirmed in 12 cases (9 papillary and 3 follicular), an incidence of 6.2%. Of these 12 cases, cytology diagnosed malignancy (papillary carcinoma) in 8; 2 nodules were cytologically suspected of being follicular carcinoma, and 2 were cytologically judged to be benign (1 Hürthle-cell adenoma and 1 adenomatous goiter). There were no cases of medullary carcinoma. The incidence of benign lesions was 93.8% (183 of 195). Of the 33 benign lesions with histologic study, cytology had diagnosed 10 follicular adenomas, 8 colloid nodules, 2 cysts of the thyroid, 9 cases of lymphocytic thyroiditis, 2 Hürthle-cell adenomas, and 2 adenomatous goiters. Surgery was deferred in 150 cases, with the patients placed on suppressive therapy or observation. The cytopathologic findings in these cases included chronic lymphocytic thyroiditis (42), colloid goiter (56), adenomatous goiter (24), thyroid cyst (19), cystic colloid goiter (4), colloid (3), and hyperplastic goiter with regression (2). TNA biopsy examination of clinically single or dominant thyroid nodules is accurate in selecting which patients should undergo surgery or only LT4 suppressive therapy or observation.

VIRGINIA MEDICAL

Obstinate

WHY do I come to these meetings? These physicians are absolutely impossible. To call them hard-headed and obstinate is the understatement of the year. They came to this meeting with their minds already made up, their thoughts set in concrete. They pay no attention when ideas contrary to their mind-set are presented, and, worse yet, they do not hesitate to interrupt anyone who has the floor. They are not about to listen to the sweet voice of reason.

We physicians are not born god-like, but we certainly acquire that attitude during our training. After all, we had to be smart to get into medical school, much less to graduate. Our employees defer to us. Our patients seek our advice even though they often do not heed it. Every day we sign all sorts of official documents so that the world will continue to spin. We make life and death decisions routinely and decisively. We must be independent thinkers.

Nonetheless, I would rather fight lions and tigers barehanded than to argue with this group of doctors. My stomach churned before the meeting started, and I am sure to leave with a splitting headache.

I am beginning to think that I will never convince all thirty of them that I am the one who is right.

H. S. CAMPELL, MD

Our Health Care System

ALMOST all the developed countries have a national health care system. Some are very good; others are completely inadequate. Barbara Ehrenreich (Time magazine, December 10, 1990) bitterly assails the American free enterprise health care system. She points out that we spend $600 billion a year on medical care (11% of GNP), yet 37 million Americans have no health insurance and are unable to financially manage even relatively minor illnesses. For their plight she places the blame squarely on the insurance industry.

Two of the annual lectures at St. Mary's Hospital in Richmond, the Bishop John J. Russell Lecture and the Sister Rita Thomas Lecture were combined last spring in a program on health care in other countries. Professor Neil McIntyre, chairman of the Department of Medicine, Royal Free Hospital, London, presented the British Health Care Services and Dr. Judith C. Kazimirski, President of the Canadian Medical Society, presented the Canadian health care system. Of the two, the British program is the older; the Canadian system is of more recent vintage, and is generally considered to be one of the more acceptable ones. While more Americans who support a national health care program seem to favor the Canadian system, the British service provides one important factor; it is permissible for private practitioners of medicine to coexist.

The cost of medical care in the United States continues to escalate; the problem of access to medical care has not been solved. The availability of health insurance through the insurance industry naturally is limited to those who are reasonably affluent. Medicare and Medicaid are inadequate and a mass of red tape. This writer believes in and supports the concept of a free enterprise health care system. However, if the free enterprise system is to endure in this country,

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inequity must be resolved. It is not necessary for every patient to be in a private hospital room, with special duty nurses in attendance, any more than it is necessary for every American to drive a Mercedes or BMW; however, inequity as far as access to good medical care must be resolved. If the insurance industry, with the help of the medical profession, is unable to accomplish this goal, national health insurance of some type will almost certainly come. And it won't be as long as it has been.

E.L.K., Jr.

Principles of Practice for the Male Physician

1. Avoid bizarre costumes and unusual hair styles. You are supposed to help your patients, not frighten them.
2. A coat and tie are a must.
3. When visiting a patient, in home or hospital, sit down.
4. Do not sit on the patient's bed.
5. Do not call patients by their first names.
6. Do not call other non-MD health-care personnel (nurses, dieticians, secretaries, technicians, etc.) by their first names.
7. Do not become intimate with your female patients.
8. No "dear," "honey," "sweetie," "darling."
9. You can't practice medicine on the telephone.

Kinloch Nelson, MD

1621 Hanover Avenue
Richmond VA 23220

CORRECTIONS

The physician in this picture was misidentified in the January issue. He is Dr. Walter W. Schroeder of Norfolk, *not* Dr. Mark T. Schreiber of Virginia Beach as the caption erroneously stated. Dr. Schroeder was photographed at the Medical Society of Virginia's annual meeting at the Homestead. In the same issue, the caption for a picture of Dr. Albert L. Roper II identified him correctly but located him in Hampton. Not so. He lives and practices in Norfolk. The Editors greatly regret.

OBITUARY

• **Woodland Ward Anderson, Jr., MD,** retired Newport News dermatologist; University of Virginia School of Medicine, 1944; age 69; died January 10, 1991, while vacationing in Key Largo, Florida.

• **James Duncan Beaton, MD,** retired Danville physician; Boston College of Physicians and Surgeons, 1942; age 75; died November 16, 1990.

• **Edward Vernon Brush, Jr., MD,** Lexington; University of Virginia School of Medicine, 1937; age 82; died January 23, 1991, after 51 years of practice.

• **Samuel Walthall Budd, Jr., MD,** Richmond internist; Yale University School of Medicine, 1942; age 75; died January 12, 1991.

• **Richard Cowley, MD,** Portsmouth general practitioner; Middlesex Hospital Medical School, 1971; age 44; died November 22, 1990.

• **William Taylor Dabney III, MD,** Richmond; Medical College of Virginia, 1954; age 72; died January 16, 1991. A retired hematologist and internist, he was professor emeritus at the Medical College of Virginia.

• **Porter Burks Echols, MD,** Lynchburg ophthalmologist; University of Virginia School of Medicine, 1925; age 90; died December 23, 1990.

• **Giles Quarles Gilmer, MD,** general practitioner in Russell County for 35 years; University of Virginia School of Medicine, 1943; age 71; died November 5, 1990. A son, Robert Dickenson Gilmer, MD, Abingdon, survives him.

• **Marion Kemper Humphries, Jr., MD,** Charlottesville; University of Virginia School of Medicine, 1937; age 80; died January 26, 1991. He had been chairman of the University of Virginia's Department of Ophthalmology.

• **Thomas E. Padgett, MD,** Portsmouth radiologist; University of Louisville School of Medicine, 1951; age 77; died October 26, 1990.

• **Gaston E. Roy, MD,** Springfield psychiatrist; Laval University Faculty of Medicine, Quebec, 1962; age 55; died March 13, 1990.

• **John Earle Smith, MD,** Richmond; Medical College of Virginia, 1950; age 67; died December 29, 1990. He had been medical director of the Masonic Home of Virginia for 35 years.

• **Weir Mitchell Tucker, MD,** retired Richmond neurologist/psychiatrist; University of Virginia School of Medicine, 1942; age 77; died February 26, 1991. He was for many years president of Tucker Hospital, a psychiatric center founded by his father, Dr. Beverley R. Tucker.

Memoir of John Collier 1906-1990

By Charles M. Caravati, MD

Dr. John Elwood Collier, long-time practitioner of family medicine in Richmond, died on April 1, 1990. Dr. Collier was born in Linden, North Carolina, and graduated from Wake Forest University in 1927 and from the Medical College of Virginia in 1931. After an internship at St. Luke's Hospital, he began the practice of medicine in association with Charles M. Caravati, with whom he continued to practice for 20 years. Ten of those years were in association with Dr. William L. Wingfield. Dr. Collier was the director of the C&O Railroad clinic and also the physician for the Hermitage Home. He retired in 1985 after spending a few years with Dr. Robert Williams and associates.

Dr. Collier was a lifetime member of the Kiwanis Club and a deacon of the Tabernacle Baptist Church. He is survived by his wife, Elizabeth Melton Collier; two daughters, Elizabeth C. Payne and Judith C. Perry, both of Richmond; one son, John E. Collier, Jr., of Roanoke; one sister, Florence C. Moye of Maury, North Carolina; and five grandchildren.

Memoir of James Kirkland 1935-1990

James Arlington Kirkland, MD, of Emporia was born in LaCrosse, Virginia, on March 4, 1935, and died unexpectedly on July 13, 1990, at the age of 55.

Dr. Kirkland received his undergraduate degree in 1956 and then graduated in 1960 from the University of Virginia School of Medicine. He began his medical practice in Jarratt, Virginia, in 1961. He later joined with two physicians in Emporia and formed the Kiser-Allison-Kirkland Clinic. In 1969 he, along with the other two physicians, joined with three other local physicians to form Emporia Medical Associates, PC. He continued to practice medicine in both Emporia and Jarratt as a family physician until 1985, when he retired from his medical practice due to his health.

He was a member of the American Academy of Family Physicians, The Medical Society of Virginia, the Fourth District Medical Society and the American Medical Association.

Dr. Kirkland served as mayor of the City of Emporia from 1972 to 1976. His other contributions to the Emporia-Greensville community included: president of the Emporia Volunteer Fire Department, advisor to the Greensville Memorial Hospital, advisor to the Greensville Volunteer Rescue Squad, past president of the Emporia Shrine Club and a member of the Widow's Son Masonic Lodge No. 150 AF & AM. He was a member of the Main Street United Methodist Church.

His death will be a great loss to the community he served so well, both as a professional and as a friend.

He is survived by his wife, Elizabeth S. Kirkland; one daughter, Carole E. Kirkland of Richmond; a son, Randolph M. Kirkland, of Emporia; his father John E. Kirkland of LaCrosse; and three brothers, William E. Kirkland of Staunton, John H. Kirkland of Boydton, and Hugh L. Kirkland of San Antonio, Texas.

Memoir of C.H. Binford 1900-1990

By Willys M. Monroe, MD

A bright star faded from the medical firmament when Chapman Binford died on February 9, 1990. Although Dr. Binford never practiced medicine in Virginia, he was a Virginian in every sense of the word, beginning with his birth in Prince Edward County on October 3, 1990. He was a lifelong member of the Medical Society of Virginia, a Phi Beta Kappa graduate of Hampden-Sydney College, Class of 1923, and a graduate of the Medical College of Virginia, Class of 1929. In the two years between college and medical school, Dr. Binford was principal of Appomattox High School. After graduation from MCV, Dr. Binford served an internship at the U.S. Marine Hospital, Norfolk. Upon completion of his internship, he was commissioned assistant surgeon, U.S. Public Health Service, and remained on duty on the medical service of the Norfolk Marine Hospital, where he came under the influence of Dr. Walter B. Martin, his medical consultant and mentor. Dr. Binford always spoke warmly of Dr. Martin as "my favorite doctor." In 1931, the U.S. Public Health Service assigned Dr. Binford to a two-year tour of duty in cancer research at Harvard Medical School; and in 1933, he was transferred to the U.S. Public Health Service leprosy station in Hawaii. This was his first exposure to the clinical aspects of this fascinating disease, for which at that time there was no effective treatment. He recorded the progress of the disease with personally taken photographs, which are classics even by today's standards. In 1936, he began a 15-year detour through pathology training at the National Institutes of Health, and a broad and rich experience in general pathology at several of the Marine Hospitals. After World War II, he established a training program in pathology at the Baltimore Marine Hospital. It was there we met in 1946, and there it was my inestimable privilege to become the first resident in his program. Our friendship continued thru the decades and his example, both personal and professional, has been a guiding light to me and to scores of other aspiring young physicians. In 1951, Dr. Binford was assigned to the Armed Forces Institute of Pathology (AFIP). There, in 37 years, he became a legend in his own time, remaining there for 28 years in a noncommissioned status after his retirement from the commissioned corps of the U.S. Public Health Service in 1960. He continued to work at his office until the fall of 1988, when increasing problems with congestive heart failure made it advisable to discontinue the long and treacherous drive to the Institute from his home in Arlington. Nonetheless, he kept in close contact with his colleagues by telephone and by their visits to his home. His last publication appears as the chapter on leprosy in the 1990 edition of Anderson's *Pathology*. Even in his 89th year, he continued to consult on cases of special merit—leprosy, fungal diseases, and some exotic tropical diseases.

At the Armed Forces Institute of Pathology, Doctor Binford served as chief of the Infectious Disease Branch ('51-'60) and as registrar of the Leprosy Registry ('51-'76) and in 1960, he established the Geographic Pathology Division and served as its first chief ('60-'63). His approach to geographic pathology is illustrated by the contacts and interactions he developed in the Third World. While chief of the Division he established research units in Uganda, South Africa, the Philippines and Thailand, and he developed liaisons with mission hospitals in many developing countries. In 1963, he stepped aside as chief of Geographic Pathology to become medical director of the Leonard Wood Memorial (leprosy foundation), but continued to serve as chief of Special Mycobacterial Disease Branch at the AFIP ('63-'76).

Until the mid-1970s the unchartered American Registry of Pathology (ARP) was the AFIP's link to civilian medicine. In 1975, a strong movement emerged at the Pentagon to eliminate the ARP and restructure the AFIP along strictly military lines. Dr. Binford worked tirelessly behind the scenes as a private citizen serving as liaison between the AFIP's professional staff and a representative of the Senate Health Subcommittee to preserve the traditional missions of the ARP and the AFIP. The ARP received a congressional charter as a result of Dr. Binford's efforts, legalizing and protecting the liaison between the AFIP and civilian medicine. He acted as executive

officer of the ARP ('77–'80). The ARP not only links military and civilian medicine but serves as a resource for teaching and research in national and international medical science. As the scope and impact of the ARP grow, Dr. Binford's foresight is appreciated.

It was Dr. Binford's long experience with the clinical and pathological aspects of leprosy that led him to hypothesize in 1956, that the leprosy bacillus has a natural preference for sites of lower body temperature. This observation led to the discovery of the first animal model for leprosy, and to the inoculation of *M leprae* into the armadillo, an animal with body temperature of 32°-35° C. Today, armadillo tissues provide the only abundant source of *M leprae* for clinical and laboratory studies. *M Leprae* from armadillos led to the Immunology of Leprosy Program (1974) sponsored by the World Health Organization. The goals of the program were the development of a vaccine and method for identifying patients with subclinical infections. Studies are now under way.

Dr. Binford was an authority on the histopathology of fungal diseases. He was coauthor of the textbook, *Medical Mycology*, now in its third edition (1777), and was author of many articles on cryptococcosis, chromomycosis, cladosporiosis, and histoplasmosis. Dr. Binford contributed chapters to and coedited (with Daniel H. Connor, MD) *The Pathology of Tropical and Extraordinary Diseases* (1976), the definitive work in this field. All in all, his publications include seven books and monographs, 31 chapters in scientific medical books, and 105 scientific articles.

In his later years, many unsought honors came to Dr. Binford. Each of his alma maters conferred honorary ScD degrees (Hampden-Sydney, 1962; MCV, 1979). He was elected to Alpha Omega Alpha and Sigma Xi. He served as president of several scientific societies. His name appears in *Who's Who in America* and *American Men and Women of Science*. He was editor of the journal *International Pathology* (1960-1965). He received the Ward Burdick Award of the American Society of Clinical Pathologists (1968); he was the Maude Abbott Lecturer, International Academy of Pathology (1973) and the Walter Reed Lecturer, Richmond Academy of Medicine (1983), to mention only a few.

CHAPMAN H. BINFORD, MD
1900-1990

Finally and most of all, I must add my personal note of Dr. Binford as a person—friend, teacher, scientist. He was astute, selfless in his promotion of his colleagues and very generous. He was tireless in fostering the projects and welfare of others. His style was to train others, then quietly step aside when they were ready for increased responsibility. When approached with ideas for research or diagnosis, he was always supportive and strove to garner the best from the suggestions of colleagues. He seemed always to be able to bring ideas into being and studies to fruition. He was always willing, indeed frequently insisted on remaining anonymous when collaborating on projects. Dr. Binford's long and distinguished career as a consultant, researcher, educator, administrator, advisor and friend will continue to be an inspiration for all of us whose privilege it was to know and work with him.

NEW MSV MEMBERS

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Willie A. Andersen, MD, Gynecological Oncology, Box 387, University of Virginia Health Sciences Center, Charlottesville VA 22908

Mett B. Ausley, Jr., MD, Pathology, Martha Jefferson Hospital, 459 Locust Avenue, Charlottesville VA 22901

David S. Duani, MD, Family Practice, PO Box 425, Stanardsville VA 22973

Richard W. McCallum, MD, Gastroenterology, Box 145, University of Virginia Health Sciences Center, Charlottesville VA 22908

E. Williams Pelton II, MD, Neurology, 400 Locust Avenue, Charlottesville VA 22901

Karen S. Rheuban, MD, Pediatric Cardiology, Box 386, University of Virginia Health Sciences Center, Charlottesville VA 22908

Brian Wispelwey, MD, Infectious Diseases, Box 397, University of Virginia Health Sciences Center, Charlottesville VA 22908

Alexandria Medical Society

Suresh K. Malhotra, MD, Gastroenterology, 4921 Seminary Road, Alexandria VA 22311

James A. Vafier, MD, Emergency Medicine, 8013 Daffodil Court, Springfield VA 22152

Chesapeake Medical Society

Scott P. Eichelberger, MD, Pediatrics, 637-D Kingsborough Square, Chesapeake VA 23320

Danville-Pittsylvania Academy of Medicine

Mark C. Hermann, MD, Orthopedic Surgery, 115 Executive Drive, Danville VA 24541

Dugan W. Maddux, MD, Nephrology, 1040 Main Street, Danville VA 24541

Franklin W. Maddux, MD, Nephrology, 1040 Main Street, Danville VA 24541

Fairfax County Medical Society

Ahmad Ellini, MD, Pediatrics, 7501 Little River Turnpike, Annandale VA 22003

Samuel O. Grimm III, MD, Obstetrics/Gynecology, 410 West Maple Avenue, Vienna VA 22180

Carol S. Schuffler, MD, Gastroenterology, 8301 Arlington Boulevard, Fairfax VA 22031

Wan Shin, MD, Physical Medicine/Rehabilitation, PO Box 7446, Fairfax Station VA 22039

Simon J. Simonian, MD, General Surgery, 3301 Woodburn Road, Annandale VA 22003

Robert B. Stinger, MD, Orthopedic Surgery, 313 Park Avenue, Falls Church VA 22046

Michael G. Velchik, MD, Nuclear Medicine, 10311 Greenwood Place, Oakton VA 22124

Robert P. Younes, MD, Pediatrics, 5109 Leesburg Pike, Falls Church VA 22041

Fredericksburg Area Medical Society

Thomas E. Martyak, MD, Cardiovascular Diseases, 1701 Fall Hill Avenue, Fredericksburg VA 22401

Lynchburg Academy of Medicine

Clinton E. Clapp, MD, Thoracic Surgery, 1900 Tate Springs Road, Lynchburg VA 24501

C. Michael Valentine, MD, Cardiology, 2215 Landover Place, Lynchburg VA 24501

Mid-Tidewater Medical Society

Michael Halpert, MD, General Surgery, PO Box 2098, Tappahannock VA 22560

Newport News Medical Society

Richard R. Edwards, MD, Cardiology, 11835 Fishing Point Drive, Newport News VA 23606

Robert E. Taylor, MD, Anesthesiology, 216-A Cedar Road, Poquoson VA 23662

Norfolk Academy of Medicine

Joseph J. Baum, MD, Family Practice, Eastern Virginia Medical School, PO Box 1980, Norfolk VA 23501

Lawrence F. Cohen, MD, Orthopedic Surgery, 6275 East Virginia Beach Boulevard, Norfolk VA 23502

Terence C. Davies, MD, Family Practice, Eastern Virginia Medical School, PO Box 1980, Norfolk VA 23501

S. Margaret Davies, MD, Family Practice, Eastern Virginia Medical School, PO Box 1980, Norfolk VA 23501

Jeffrey A. Klein, MD, Diagnostic Radiology, 1133 Chumley Road, Virginia Beach VA 23451 *Cont'd on page 125*

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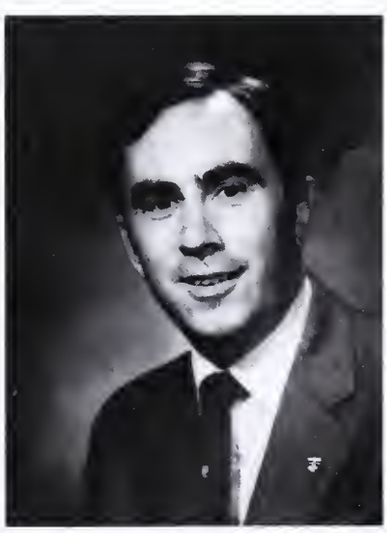

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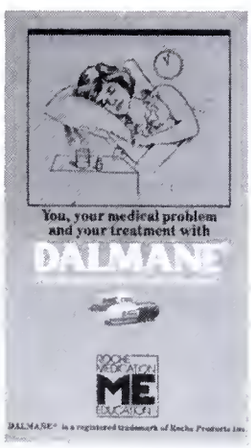

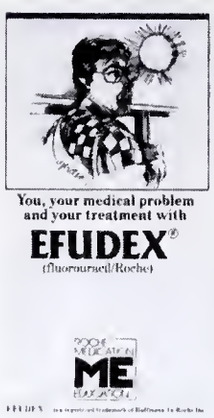

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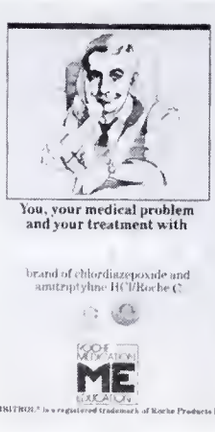

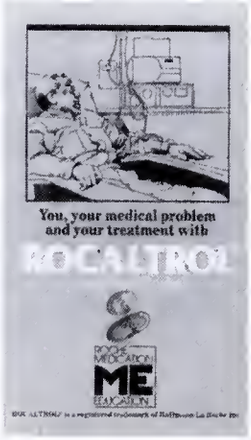

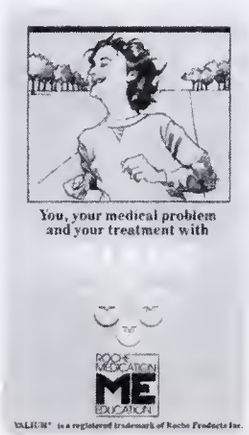

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Northern Virginia Medical Society
Cyril A. Barch, MD, Internal Medicine, 1870 Amherst Street, Winchester VA 22601

Patrick Henry Medical Society
David W. Seamon, MD, Pediatrics, PO Box 5351, Martinsville VA 24115

Portsmouth Academy of Medicine
Jose M. DeJesus, MD, Anesthesiology, 415 West York Street, Norfolk VA 23510
Martha W. Luckenbach, MD, Ophthalmology, 3101 American Legion Road, Chesapeake VA 23321

Prince William County Medical Society
Elizabeth A. Roseberry, MD, Neonatal/Perinatal Medicine, PO Box 610, Occoquan VA 22125
John D. Wall, MD, Obstetrics/Gynecology, 9001 Digges Road, Manassas VA 22110

Richmond Academy of Medicine
Mark R. Bladergroen, MD, Cardiovascular Surgery, 7107 Jahnke Road, Richmond VA 23225
Randall E. Dalton, MD, Otolaryngology, 505 West Leigh Street, Richmond VA 23220
Susan S. Park, MD, Obstetrics/Gynecology, 5855 Bremo Road, Richmond VA 23226
Edmund D. Schoeffler, MD, Obstetrics/Gynecology, 5855 Bremo Road, Richmond VA 23226
Lorraine J. Trow, MD, Pulmonary Diseases, 5855 Bremo Road, Richmond VA 23226
Marie E. Tummillo, MD, Obstetrics/Gynecology, 5855 Bremo Road, Richmond VA 23226
Timothy J. Zimmer, MD, Orthopedic Surgery, 8919 Three Chopt Road, Richmond VA 23229

Roanoke Valley Academy of Medicine
J. Samuel Mitchener III, MD, Plastic Surgery, 117 McClanahan Street, Roanoke VA 24014

Rockbridge County Medical Society
Frank W. Price, MD, Pediatrics, Route 7, Box 90, Lexington VA 24450

Southside Virginia Medical Society
Paul H. Laughlin, MD, Emergency Medicine, 101 Gateway Centre Parkway, Richmond VA 23235

Southwestern Virginia Medical Society
Kenneth G. Gilbert, MD, Psychiatry, 404 Washington Street, Galax VA 24333
Margaret J. Gregorczyk, MD, General Practice, Route 3, Box 5, Rosedale VA 24280
David C. Hadden, MD, Orthopedic Surgery, 1154 Snider Street, Marion VA 24354
Susan G. Mourot, MD, Family Practice, 710 West Ridge Road, Wytheville VA 24382

Stuart Medical Society
William H. Boyce, MD, Urology, Route 2, Box 250, Stuart VA 24171

Tri-County Medical Society
Bartholomew M. LoPresti, MD, General Practice, 919 South Church Street, Smithfield, VA 23430

Virginia Beach Medical Society
Jorge B. Joaquino, MD, General Practice, 1488 Five Forks Road, Virginia Beach VA 23455
Octavian C. Tomescu, MD, General Surgery, 1856 Colonial Medical Court, Virginia Beach VA 23454

Williamsburg-James City County Medical Society
Philip S. Walter, MD, Internal Medicine, 230 Monticello Avenue, Williamsburg VA 23185

Residents
Reid B. Adams, MD, General Surgery, 687 Victorian Court, Charlottesville VA 22901
Sylvia S. Hendrix, MD, Radiation Oncology, Box 383, University of Virginia Health Sciences Center, Charlottesville VA 22908
Carole L. Light, MD, Internal Medicine, Route 1, Box 137, Clifton Forge VA 24422
Darya M. Maanavi, MD, Obstetrics/Gynecology, 2230 George Marshall Drive, Falls Church VA 22043
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